UNIVERSITY OF LIVERPOOL

Exploring Gustatory Neural Coding and the Influence of Appetite and Expectancy

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Thesis submitted in accordance with the requirements of the University of Liverpool for the degree of Doctor in Philosophy by Moon Wilton

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Declaration

No portion of this work has been submitted in support of any other application for degree or qualification at this or any other University or institute of learning.

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List of Abbreviations

AI – Anterior Insula

Area G – Gustatory area

BOLD - Blood-Oxygen-Level Dependent

BMI – Body Mass Index

CRT – Cathode Ray Tube

db – Decibels

dlPFC – Dorsolateral Prefrontal Cortex

EEG – Electroencephalography

EMG - Electromyography

ERD/S – Event-Related De/Synchronisation

ERP – Event-Related Potential

fMRI – functional Magnetic Resonance Imaging

FO – Frontal Operculum

gERP – gustatory Event-Related Potential

gLMS – general Labelled Magnitude Scale

GMP – Guanosine 5′-Monophosphate

HCl – Hydrochloride

ISI – Inter-Stimulus Interval

LPP – Late Positive Potential

LMS – Labelled Magnitude Scale

M – Molar

MEG – Magnetoencephalography

MF – Magnetic Field

mM – Micro-Molar

ms – Milliseconds

MSG – Monosodium Glutamate

MVPA – Multivariate Pattern Analysis

N – Negative

NaCl – Sodium Chloride

NTS - Nucleus of the Solitary Tract

OFC - Orbitofrontal Cortex

P – Positive

PGC – Primary Gustatory Cortex

POP – Parietal Operculum

prACC - pregenual Anterior Cingulated Cortex

PROP – 6-n-propythioracil

PTC – Phenylthiocarbamide

TFR – Time Frequency Representation

tt – Two recessive alleles

Tt – One dominant and one recessive allele

TT – Two dominant alleles

VAS – Visual Analogue Scales

vlPFC - Ventrolateral Prefontal Cortex

VPM - Ventroposterior Medial Nucleus

QHCl – Quinine Hydrochloride

Abstract

The purpose of this thesis was to explore human gustatory processing and how it is influenced by appetite and expectancy. The initial two years of the doctorate were dedicated to developing a gustometer mechanism and taste stimulus set to employ in the experimental investigations. Event-related potentials (ERPs), source-localised ERPs and event-related de-synchronisations and synchronisations (ERD/S) were then evaluated in response to taste characteristics under a variety of conditions.

The first experiment assessed the ERP, source-localisation and ERD/S components associated with the processing of taste quality (sweet, salt, bitter, water), intensity (neutral, weak, medium, strong) and hedonicity (pleasant, unpleasant, neutral). Gustatory stimulation evoked activations within the primary gustatory cortex (PGC) and intensity was represented in early ERP epochs and by alpha- and beta-band ERD. Hedonicity was coded in late ERP epochs and by alpha-band ERD. Taste quality coding was difficult to determine from the EEG data. The second experiment compared the processing of pleasant sweet and unpleasant bitter tastes during states of hunger following overnight fasting and satiety induced by a standardised liquid meal. Hunger and satiety evoked maximal responses to tastes from limbic regions. Hunger greatly enhanced ERP and beta-band ERS responses to tastes in general. However, responses to sweet tastes were dependent on hunger state; with enhanced neural signals in response to sweet taste after satiating on a sweet meal - suggesting differential attentional and evaluative mechanisms employed under fasted and fed conditions. A final experiment examined the influence of cue-elicited expectancy on the processing of sweet tastes. Participants were validly or invalidly cued to expect a low- or high-concentration of sweet taste; both behavioural and neural responses to invalidly cued tastes assimilated to those that were produced by the taste the participants were cued to receive. These effects began ~100 ms after the onset of the tastes, suggesting that expectancy influences the early perceptual processing of taste.

The overall findings of this thesis provide some of the first accounts of the temporal, source-localised and oscillatory dynamics of gustatory coding. The results also provide important implications for understanding how people's experience of taste and food can be modified by appetite and expectancy.

Chapter One:
General Introduction

1.1.Taste Perception

Taste is the main sensory modality by which food is evaluated. In normal human consumption, all food and drink sources pass through the oral cavity, providing a universal location for sensing and evaluating what should be ingested or rejected (Breslin & Huang, 2006). Ingested substances prompt other taste-cued reflexes, such as exocrine (e.g., gastric acid) and endocrine (e.g., insulin) secretions that anticipate and facilitate digestive processes and are necessary for normal digestion to take place (e.g., Katschinski, 2000). Without taste perception people often do not eat without medical intervention, as is observed in cancer patients who experience a radiation-induced loss of taste (e.g., Kokal, 1985; Maes et al., 2002). As such, taste is arguably the most crucial sensory modality for survival (Breslin & Huang, 2006).

The development of human taste perception is considered to be related to evolutionary trends and feeding strategies (e.g., Frank, Thomas & Hettinger, 1992; Hladik, Pasquet & Simmen, 2002). Research suggests that each species taste system is tuned to the food sources for which it is specialised (e.g., Hellekant, Glaser, Brouwer & van der Wel, 1981). For instance, bitter taste perception varies among primate species in relation to the potential toxicity of plants in their specific environment (Simmen, 1994). Moreover, humans, non-human primates and rodents all show a marked preference for sucrose corresponding with a high energy feeding strategy and the widespread occurrence of sugars in plants (Breslin & Spector, 2008; Drewnowski, 1997; Frank et al., 1992). Although it is argued that sugars are the least essential of all nutrients, containing less metabolisable energy than that of protein (Johnson, Willson, Thompson & Bertin, 1985; Livesey & Elia, 1988), the universality of sweet taste preference does suggest that sweet taste at least signifies edibility, if not nutritional value (see Ramirez, 1990, for a review). Likewise, sensitivity to salt is argued to have evolved to facilitate the maintenance of osmotic

body balance (Beuchamp & Cowart, 1985; Hladik et al., 2002; Mela & Catt, 1996). However, in modern man's ecological niche, salt palatability and intake in humans occurs in a 'need free' state and sensitivity to salt is not inversely related with its availability in the body (Mattes, 1997).

Whatever adaptive pressures determined the specifics of human taste perception, the system has evolved to rapidly detect and evaluate substances for consumption or rejection. This complex mechanism must code the fundamental characteristics of the tastant; its quality, intensity and hedonicity (e.g., Breslin & Spector, 2008; Lundy, 2008; Mattes, 1985; Moskowitz, 1977; Small et al., 1997; Small et al., 2006; Smith & St John, 1999) amongst a background of other information sources such as current appetite (e.g., Berridge, 1991; Cabanac, 1971; Drobes et al., 2001; Hasse, Cerf-Ducastel & Murphy, 2009; Mauler, Hamm, Weike, & Tuschen-Caffier, 2006; Rolls et al., 1981), information from other sensory modalities (Cliff & Noble, 1990; Frank, Hettinger, & Mott, 1992; Frank et al., 1988; Schifferstein & Verlegh, 1996) as well psychological information relating to prior experience and expectations (e.g., DuBose, Cardello & Maller, 1980; Johnson & Clydesdale, 1982; Hyman, 1983). This combination of information is instantaneously evaluated by our gustatory system allowing for quick decisions on food selection.

1.2. Taste Coding

1.2.1. Peripheral taste mechanisms

Taste is detected when soluble substances react with peripheral receptors located on the upper surface of the tongue and soft palate, and on the epiglottis and pharynx (e.g., Breslin & Spector, 2008). Specifically, these receptors extend their microvilli through the apical pore, which provides a surface for the binding of taste stimuli and the facilitation of taste transduction (e.g., Breslin & Spector, 2008). Peripheral taste receptors are innervated by primary sensory axons that run in specific branches of the chorda tympani, glossopharyngeal and vagus nerves (Witt, Reutter & Miller, 2003). Trigeminal neurons also surround taste buds, but have no synaptic contact (Getchell et al., 1991; Whiteneah, Beeman & Kinsella, 1985). Their role in taste perception is unclear; however they are thought to be involved in thermal taste (Green & Lawless, 1985; Jacobs et al., 2002; Silver & Finger, 1991) and

somatosensation (Beidler, 1953, 1965; Bryant & Moore, 1995; Kosar & Schwartz, 1990; Lundy & Contreras, 1994; Pittman & Contreras, 1998; Sostman & Simon, 1991; Wang, Singhvi, Kong & Scott, 1993).

1.2.2. Central taste mechanisms

Taste information from the cranial nerves terminates in the primary visceral sensory nucleus of the brainstem - the nucleus of the solitary tract (NTS) of the medulla (Torvik, 1955). The NTS, in turn, projects to ventroposterior medial nucleus (VPM) of the thalamus (Beckstead, Morse & Norgren, 1980). From here, fibres project to the primary gustatory cortex (PGC), which is considered to be located in the insula cortex and overlying opercula (de Araujo et al., 2003a; de Araujo et al., 2003b; Frey & Petrides, 1999; Iannilli et al., 2012; Ogawa et al., 2005; Ohla, Toepel, le Coutre & Hudry, 2010; Schoenfeld et al., 2004; Seo et al., 2011; Small et al., 1997; Veldhuizen, Bender, Constable & Small, 2007), areas also involved in orofacial somatosensory processing (Burton, Videen & Raichle, 1993; Friedman et al., 1986; Schneider et al., 1993) and olfaction (Scott & Plata-Salaman, 1999). Some studies have also implicated the transition between the parietal operculum (POP) and insula as part of the PGC (e.g. Iannilli, Noening, Hummel & Schoenfeld, 2014; Kobayakawa et al., 1996, 1999; Mizoguchi et al., 2002), a region corresponding to area G (gustatory area) in non-human primates (Kobayakawa et al., 1996).

Meta-analyses of gustatory imaging studies have identified a number of regions responsive to taste stimuli; including bilateral insula and overlying operculum, left lateral orbitofrontal cortex (OFC), right medial OFC, the pregenual cingulated cortex (prACC), and right mediodorsal thalamus (Small, 2010; Veldhuizen et al., 2011). However, these analyses failed to precisely localise the PGC and thus there is no clear description of the region and what specific taste functions these areas regulate. Although there are a number of potential reasons that could explain the variability in regions activated by taste stimuli (including variations in the deliverance of tastants and imaging methods), one explanation could be that functional specialization exists and the use of different tasks and stimuli results in differential recruitment of specialised regions or networks (Ianilli et al., 2014; Small et al., 2007).

Evidence of projections from PGC regions to several regions of the OFC has implicated the OFC as a secondary gustatory cortex (Baylis, Rolls & Baylis, 1995;

Carmichael & Price, 1995; Powell, Cowan & Raisman, 1965). Within the OFC exist taste responsive neurons (Rolls, Yaxley, & Sienkiewicz, 1990); however, many of the cells in the OFC are heteromodal (respond to many sensory modalities); with only a small proportion (20%) of cells that respond to tastes (Pritchard, 2005). This responsivity differs from that of sensory cortex cells in other modalities, which are mostly unimodal (respond to a single sensory modality; e.g., Phillips & Irvine, 1981). Nevertheless, stimulus specific responses to pure tastes have been observed in the OFC (O'Doherty et al., 2001; Small et al., 1997, 2003; Zald, Lee, Fleugel & Pardo, 1998; Zald, Mattson & Pardo, 2002). Bitter tastes have been shown to activate a region of the left anterior OFC (Chiavaras & Petrides, 2000), whereas sweet tastes have been shown to activate the right caudalateral OFC (Small et al., 2003; Zald et al., 2002). Given these findings, along with the known connections between the OFC and the ventral lateral prefrontal cortex (vlPFC), a region strongly associated with cognitive processes (see Carmichael & Price, 1996); it has been proposed that the OFC may be involved with the cognitive and affective aspects of gustation (O'Doherty et al., 2007; Small et al., 1999; 2007).

Beyond the OFC, the anatomy of the taste pathway is less well defined. Taste axons have been found in the hypothalamus and amygdala, which are known to receive projections from the OFC and insula (Aggleton et al., 1980; Burton, Rolls, & Mora, 1976; Cavada et al., 2000; Mufson et al., 1981; Rolls, 1986). The lateral hypothalamic area of the macaque has been found to contain taste responsive neurons that may be modulated in a sensory specific manner to satiety (Burton et al., 1976; Rolls, 1986). Other studies have found that ~7% of primate amygdaloid neurons are taste responsive (e.g., Scott et al., 1993), although they lack discriminative capacity (Rolls & Scott, 2003). A schematic representation of the proposed gustatory pathway is given in Figure 1.1 (Simon et al., 2006).

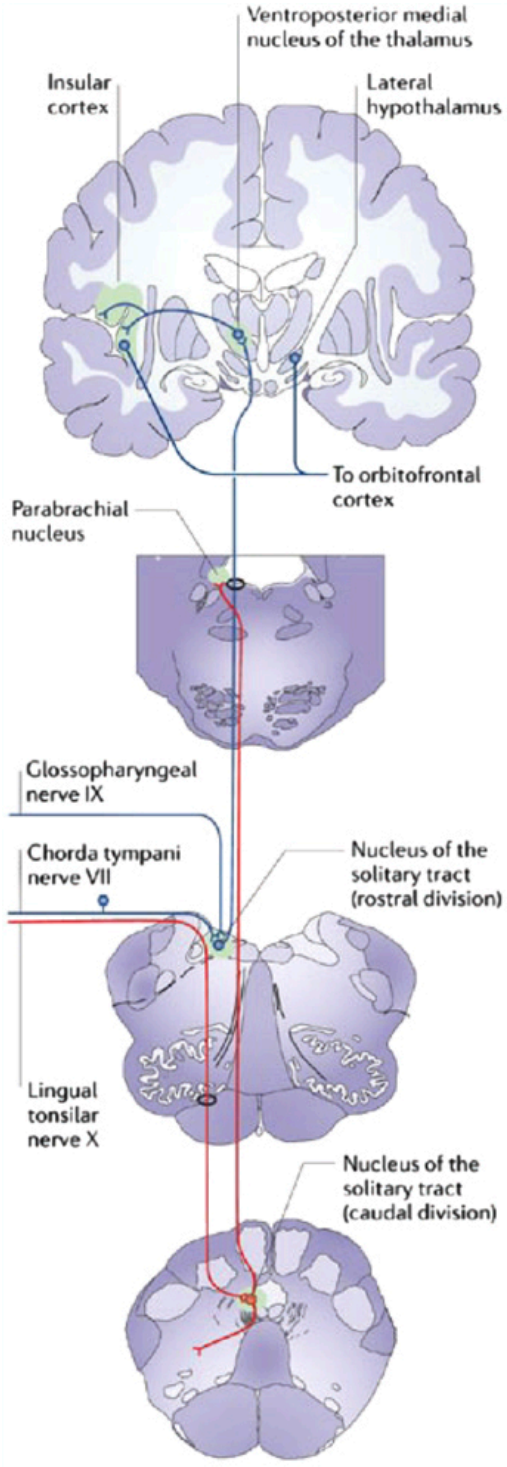

Fig 1.1. A schematic representation of the gustatory pathway adapted from Simon et al. (2006).

1.2.3. Temporal taste coding mechanisms

It has become apparent that most, if not all sensory systems utilise temporal parameters to convey information about stimuli (see Lestienne, 2001, for a review). Environmental stimuli are not static, rather they are temporally extensive and their time-varying attributes are shaped by the behavioural responses they engender (Katz, 2005). In a similar vein, neural activity is intrinsically time-varying, with brain systems transforming and communicating information over time. For instance, neural oscillations have been correlated with olfactory discrimination in moths (MacLeod et al, 1998) and have been shown to be necessary for such discriminations to take place (Nusser et al., 2001; Stopfer et al., 1997). In the gustatory system, there is evidence for functional feedback and convergence in forebrain and brainstem relays (Lundy & Norgren, 2004; Smith & Li, 2000). Thus, it is logical to assume that taste coding may be represented temporally, in that the distribution of neural activity over time may contain information about the tastant presented (e.g., Hallock & Di Lorenzo, 2006).

While there is a large volume of literature regarding the spatial localisation of the taste pathway, less is known about when this processing takes place and at what stage specific information is significant. By examining temporal coding mechanisms it is possible to determine the functional timeline of taste processing and with the use of implanted electrodes in animals (e.g., Sadacca, Rothwax & Katz, 2012) or electro-encephalography (EEG) source-localisation in humans (e.g., Ohla et al., 2010), it is also possible to locate the regions within which this information is processed; providing a means with which to complement and enhance knowledge gained from other imaging tools.

In humans, the temporal coding of sensory stimuli can be observed non-invasively using EEG. ERP analyses determine the amplitude of brain responses time-locked to the onset of an event and these events can be source-localised to estimate the region from which they originate. Moreover, ERD/S analyses can determine increases and decreases in amplitudes across functionally distinct frequency bands.

The physiological basis and functionally distinct components of the EEG signal are discussed in detail - with diagrams provided - in Chapter Two [Section 2.5, Figure 2.6 (a, b)]. Briefly, the ERP signal is categorised by its polarity [positive

(P) or negative (N) amplitude] and by the timing of its peak. For instance a P100, or P1, is a positive amplitude peak at ~100 ms after stimulus onset, or the first positive amplitude peak in the data. An N170, or N1 component would refer to a negative amplitude deflection at 170 ms after stimulus onset, or the first negative amplitude deflection within the data. Generally speaking, early ERP deflections (exogenous: < 100 ms) are associated with the processing of primary sensory characteristics; components between 100 – 300 ms (mesogenous) are linked with the convergence of sensory and attentional information; while later deflections (endogenous: > 300 ms) are linked with higher-order processing [Frank, Contreras & Hettinger, 1983; Goldstein, 2009; Hellekant et al., 1981; Katz, Simon, & Nicolelis, 2001; MacDonald, Meck & Simon, 2012; Ogawa, Hayma & Ito, 1984; Scott & Plata-Salaman, 1999: see Chapter Two, Figure 2.6 (a)].

The ERD/S signal, on the other hand, is characterised by the increase (ERS) or decrease (ERD) in the amplitude of neural oscillations within specific frequency bands (e.g., delta, theta, alpha, beta, gamma). ERS is generally associated with a deactivation of cortical areas (except in the case of theta-band oscillations), whereas ERD relates to an activated cortical area with increased excitability (Pfurtscheller, 2001). The frequency bands are linked with distinct functions. For example, theta is associated with memory processes (Klimesch, Schimke & Schwaiger, 1994), alpha with attentional mechanisms (e.g., Klimesch, 2012) and beta with motor activity [Pfurtscheller & Lopes da Silva, 1999: see Chapter Two, Figure 2.6 (b)].

Historically, very few gustatory EEG studies have taken place, owing largely to the difficulties in delivering taste stimuli with the temporal accuracy required for electrophysiological analysis. In order to examine gustatory ERPs (gERPs), a gustometer mechanism is required to present taste stimuli that can simultaneously trigger EEG data at millisecond temporal precision, in order to achieve a good summation of the evoked potentials over trials (Ohla, 2012). Moreover, the tastant must provide a strong enough percept (high enough concentration) in order to evoke neural changes, requiring rinses between each tastant and long inter-stimulus intervals (ISIs) in order to avoid habituation and adaptation (Mizoguchi et al., 2002), which is not an issue with stimulation from other senses (besides olfaction), or for the fewer trials required in functional Magnetic Resonance Imaging (fMRI). As a result, gustatory EEG studies often require unfeasible testing durations along with sophisticated equipment and programs to be able to control for the aforementioned

variables. Until recently (e.g., Crouzet, Busch & Ohla, 2015; Kobayakawa et al., 1996, 1999; Iannilli et al., 2012; Onoda et al., 2005; Singh, Ianilli & Hummel, 2011; Singh, Chhotaray & Gradas, 2015), such techniques were unavailable, meaning that the gERP and the gustatory ERD/S is yet to be well characterised.

Of the limited studies investigating gustatory temporal coding, a number of ERP, source-localised ERPs and ERD/S components have been reported. These components are discussed in detail later with reference to the taste characteristic they are purported to represent. In brief, early sensory ERP deflections to tastes have been observed. P1 peaks have been reported as early as 70 ms from tastant onset (e.g., Ohla et al., 2010) with peak latencies around 130 – 150 ms for salt (Mizoguchi et al., 2002; Wada, 2004), glucose (Wada, 2005) and electric tastes (Ohla et al., 2009, Ohla et al., 2010). These early components have been localised to the transition between the anterior insula (AI) and POP (Mizoguchi et al., 2002) and from bilateral insula, opercula cortices and the OFC (Crouzet et al., 2015; Ohla et al., 2009; Ohla et al., 2010); corresponding with areas reported as gustatory cortices in Magnetoencephalography (MEG) studies (e.g., Kobayakawa et al., 1996; 2012). N1 peaks ~ 200 ms have also been identified for electric (Ohla et al., 2009, Ohla et al., 2010) and salt tastes (Mizoguchi et al., 2002) and have been localised to the bilateral insula. Late potentials have also been observed (e.g., the late positive potential, LPP) and estimated to originate from the POP (Ianilli et al., 2014).

Gustatory ERD/S investigations are even sparser. However, it has been reported that infant theta-band ERS and alpha-band ERD occur in the right-hemisphere in the presence of unpleasant taste stimuli, and in the left-hemisphere in response to pleasant taste stimuli (Fox & Davidson, 1986). Gum chewing has been shown to induce alpha-band ERS and beta-band ERD, while adding flavour evoked alpha-band ERD and beta-band ERS (Morinushi et al., 2000). Additionally, right-frontal theta-band ERD has been observed for healthy people in response to bitter tastes and in anorexic patients in response to sweet tastes (Tóth et al., 2004).

In sum, while there are very few gustatory EEG studies to date and the morphometry of the gustatory EEG signal is yet to be characterised, there is convincing evidence to suggest that gustatory ERPs and ERD/S can be obtained. These can begin as early as 70 ms after the onset of the tastant (e.g., Ohla, 2010) and

likely originate in insula and POP regions. However, the specific information these components process is unclear.

1.3. Taste quality

1.3.1. Basic tastes

Psychophysicists have tended to divide taste into four simple qualitative categories: sweet, sour, salty and bitter, with umami often considered a fifth (e.g., Lawless, 1987). These tastes correspond somewhat with particular classes of biologically relevant compounds. For instance, the perception of sweet taste can be associated with the presence of simple carbohydrates, whereas the presence of salt taste is associated with the presence of sodium and other ions (Breslin & Spector, 2008; Pfaffman, 1976). For some time it was believed that the tongue consisted of discrete regions responding to these different qualities (Hanig, 1901), and that the gustatory cortex was organised in a similar manner (Pfaffman, 1974).

However, while bitterness, sourness, sweetness and saltiness are semantic descriptions common across human societies, they have no definitive physical basis. For instance, certain concentrations of salt have a sweet quality and not all tastes (e.g., liquorice) can be associated with these categories (Faurion, 1993). Moreover, people experience a variety of taste sensations, including fat, starch, temperature, astringency, pungency, and various metallic tastes (to name but a few). None of these, however, fit into these four categories (Purves et al., 2001) and some have recently been described as being a taste quality in their own right (e.g., Lapis, Penner & Lim, 2016; Running, Craig & Mattes, 2015). Importantly, it is now known that each taste modality can all be perceived on all tongue loci where there are taste receptors (Bartoshuk, 1993; Collings, 1974) and that there are no taste neurons specific to these the categories. Rather, specific classes of neurons respond ‘best’ to specific taste qualities (Baylis & Gaffan, 1991; Norgren & Pfaffman, 1975; Ogawa et al., 1984; Rolls et al., 1990; Scott et al., 1986; Scott & Plata-Salaman, 1999). However, defining taste in such groups has provided a useful basis for much research, and given the limited number of semantic descriptors worldwide, the familiar terms of sweet, sour, salty and bitter remain central to gustatory investigations.

1.3.2. Models of taste quality coding

Two models have been proposed to explain the neural coding of taste quality; the labelled line theory (Pfaffman, 1974) and the across neuron-pattern theory (Erickson, 1963; Erikson, Doetsch, & Marshall, 1965; Pfaffman, 1959). The labelled line theory proposes that taste quality is processed through feed-forward circuitry from peripheral receptors to higher-order coding mechanisms. According to this view, activity in a dedicated subset of neurons is sufficient and necessary to lead to the generation of a qualitative taste (e.g., Pfaffman, 1974). By contrast, the across-fibre pattern models propose that taste neurons are broadly tuned. Accordingly, they suggest that taste quality is represented by the pattern of activity across a large group of neurons and that unique combinatory patterns signify stimulus identity (e.g., Erikson, 1963).

Evidence for the former view has been largely obtained from animal investigations (e.g., Danilova et al., 2003; Danilova & Hellekant, 2004; Hellekant, Ninomiya, & Danilova, 1993). Non-human primate studies have shown that peripheral (Danilova & Hellekant, 2004) as well as chorda-tympani and glossopharyngeal responses (Danilova et al., 2003; Hellekant et al., 1993) contain fibres that respond almost exclusively to one tastant quality. However, functional studies using electrophysiology and calcium imaging in rats have shown that peripheral fibres are broadly sensitive to different taste qualities, with some cells responsive to both sweet and bitter stimuli (Caicedo, Kim & Roper, 2002; Gilbertson et al., 2001; Sato & Beidler, 2001) or both sweet and umami stimuli (Damak et al., 2003; Maruyama et al., 2006). Moreover, new evidence is emerging which suggests that peripheral taste fibres communicate with neighbouring populations and that further cells are responsible for conveying that information to afferent nerves (DeFazio et al., 2006; Kaya et al., 2004; Zhao et al., 2005). This evidence raises the possibility that information from taste cells converges within the periphery for transmission to the brain (DeFazio et al., 2006). Thus, the theory that distinct neurons generate individual taste quality representations cannot be reconciled with the data.

Within central processing mechanisms, primate studies have shown that the PGC comprise cells organised to four prototypical tastants: with ~38% most responsive to glucose, ~34% to sodium chloride (NaCl) stimuli, ~22% to quinine ~5% orientated toward the detection of hydrochloride (HCl) (Scott & Plata-Salaman,

1999). However, these cells generally respond to more than one taste quality. In rats, gustatory neurons that are strongly responsive to sweet or bitter tastants also display sensitivity to salty and acidic solutions (Di Lorenzo, Lemon & Reich, 2003; Lemon & Smith, 2005; Scott & Giza, 1990; St John & Spector, 1998; Verhagen, Giza & Scott, 2003). In addition, gustatory forebrain neurons possess descending afferent fibres that converge in the NTS and these have been shown to modulate taste activity within the brainstem (e.g., Di Lorenzo & Monroe, 1995; Smith, Li & Cho, 2005; Tokita et al., 2004). These data suggest that while there may be some segregation between taste qualities during processing, with neurons that respond 'best' to certain tastes (Frank, 1983), the system seems to be somewhat broadly tuned and continuously updated via reciprocal feed-forward and feedback mechanisms. This evidence is more in line with an across pattern view of taste coding (Erickson, 1963; Erikson et al., 1965; Pfaffman, 1959) and the idea that tastes are not discrete categories of four or five qualities (Hladik & Simmon, 1996). Although, it is possible that across patterns and labelled lines co-exist (Crouzet, Busch & Ohla, 2015).

1.3.3. Temporal coding mechanisms for taste quality

Of the limited investigations examining human temporal coding mechanisms for taste quality, some important results have been yielded. Franken et al. (2011) compared ERP responses to sucrose and artificial saliva and found that P1 and P300 amplitudes in the right hemisphere were significantly greater for sweet tastes. However, this study did not control, or measure taste intensity or hedonicity. While the P1 effects were reported to be due to the differences in taste quality, it is possible that they reflect differences in perceived intensity or affective value.

Singh, Iannilli & Hummel (2011) compared ERP responses to weak and strong monosodium glutamate (MSG) with responses to weak and strong NaCl. Results showed that P1N1 and N1P2 slopes were significantly greater for NaCl stimulation than that of MSG, independent of intensity. Topographical differences were also reported, with greater responses for NaCl observed in the parietal and frontal cortex compared with greater responses to MSG in the parietal and central cortex, suggesting a temporal as well as spatial segregation of responses to taste qualities. It should be noted, however, that despite no effects of intensity *per se*, the participant's ratings did not differ between concentrations, suggesting that they were

perceived similarly. Moreover, no hedonic measures were taken, so the effect of pleasantness on ERP amplitudes or topographic locations cannot be ruled out.

In a recent EEG study, Crouzet et al. (2015) performed a Multivariate Pattern Analysis (MVPA) to disentangle taste quality coding from the coding of other features of taste. MVPA draws information from the topographical pattern of single trials in single subjects, allowing for the direct association of single-trial brain responses with subsequent behaviour. In this study, participants tasted salt, sweet, sour and bitter solutions, reported to be of equal pleasantness and intensity, and were asked to evaluate the quality of the taste. The authors reported that taste quality coding began at 150 ms (P1) after stimulus onset with signal increases for bitter tastes, followed by salt (152 ms), sour (190 ms) and sweet (270 ms) tastes. However, individual electrodes within a given region did not provide information to discriminate between the taste qualities, suggesting that the system may utilise both spatial and temporal coding to distinguish between taste qualities.

To test this, the authors (Crouzet et al., 2015) also examined the cortical generators of the ERP responses. They found that areas associated with taste and taste quality processing (e.g., anterior and mid insula, overlying frontal and parietal operculum, superior temporal gyrus, and cuneus; e.g., Bender et al., 2009; Kobayakawa et al., 1999; Pritchard et al., 1999; Schoenfeld et al., 2004; Small, 2010) were activated by the tastes. However, there were no differences between the individual taste responses in these regions and this may need to be further examined using more spatially precise imaging methods (Crouzet et al., 2015).

The Crouzet et al. (2015) study provides a novel account of taste quality coding and goes someway to distinguishing responses from those generated by the intensity or pleasantness of the tastes. However, while the authors report little variation between the intensity (range 46 – 63 on a 100-point visual analogue scale) and pleasantness (range: 34.6 – 62.9 on a 100-point visual analogue scale) ratings; no direct comparisons were reported. Therefore, tastes rated as 34.6 for pleasantness, for example, may be significantly less pleasant that those rated 62.9 and this may have affected neuronal responses, although no correlations between EEG responses and ratings were reported (Crouzet et al., 2015). Further research directly comparing EEG responses to quality, intensity and hedonicity responses would shed more light on this mechanism.

1.4. Taste Intensity

1.4.1. Taste intensity judgements

Taste intensity refers to the magnitude at which an organism perceives a taste and is associated with the concentration of a given tastant. Although related, taste intensity perception differs from taste sensitivity, which concerns the ability to detect the presence or absence of a taste. Taste sensitivity and intensity perception are important for food selection as they act as indictors of edibility and palatability (Frank, 2005). For instance, threshold concentrations for substances that humans require for energy are quite high [i.e., sucrose, 20 millimolar (mM)], promoting greater intake, whereas thresholds for toxic substances (i.e., bitter quinine, 0.008 mM) are low, promoting detectability of potentially dangerous compounds (Purves, 2001).

Early studies of taste intensity judgements focused on detection thresholds (e.g., Harris & Kulmas, 1949). These methods were based on Fechner's (1860) classic principle of psychophysics whereby the magnitude of a subjective sensation must be proportional to the logarithm of the stimulus intensity. In early taste threshold studies (e.g., Dixon & Massey, 1969; Harris & Kalmas, 1949) subjects were presented with a stimulus that was too weak to detect followed by increasing concentrations until the subject reported a sensation (absolute threshold). An alternative measure of taste detection determines the ability of a subject to discriminate the smallest variation of taste concentration from a reference solution (Weber ratio; Weber, 1834). Participants are provided with a pair of tastants (one is a reference) and asked to select the strongest concentration. The detection threshold is the minimal detectable change, or just noticeable difference that is perceived by 50 % of subjects (Galanter, 1962; Johansson et al., 1973; Laing et al., 1993). These methods provide a useful measure of taste sensitivity and have been utilised to quantify general detection thresholds within species, specific classes of tastants and amongst those with disorders of gustatory function. However, these methods do not provide good estimates of psychophysical changes in taste intensity (see Bartoshuk, 1978, for a review).

In many sensory systems the ability to detect changes in stimuli intensity is described by the ratio of the magnitude of change, or the per cent increase or decrease in the perceived intensity of stimuli (Stevens, 1975). Magnitude estimations

direct subjects to assign numbers on a line to sensations such that one sensation that is twice as strong as another receives twice the numerical value (Stevens, 1956). Stevens (1956, 1957) devised this method to produce scales with ratio properties that could be used as a method to determine the rate at which an individual's perceived intensity grew with increases in stimulus concentration. Accordingly, the rate at which perceived intensity grows with concentration is the exponent of Steven's power function, $I = kC^n$, where I is intensity and C is concentration. Borg (1961) adapted this methodology so it could be utilised to compare across subjects and groups. To do this, he added verbal descriptors to the line scale ranging from 'no sensation' to 'maximum sensation', with intermediate labels such as 'weak and 'very strong', although the location of the verbal anchors were not empirically deduced.

Building on this, Green et al. (1993) developed the Labelled Magnitude Scale (LMS) designed specifically for the measurement of oral sensations. They asked 33 subjects to provide magnitude estimates for a variety of oral experiences, along with estimates for intensity descriptors (e.g., 'weak', 'strong') and required subjects to locate the top of the scale. The resulting rating scale is composed of seven verbal labels arranged according to the geometric means of their rated magnitudes (Green et al., 1996). A key feature of the LMS is the quasi-logarithmic spacing of the verbal labels along a single line and the inclusion of 'strongest imaginable' (referring to any oral experience) as its uppermost descriptor and 'barely detectable' as its lowermost (intermediate labels are 'weak', 'moderate', and 'strong'). Thus, the LMS provides a direct, user-friendly scaling method to represent perceived sensory magnitude (e.g., Lawless et al., 2010).

The LMS was later adapted by Bartoshuk et al. (2004). It was felt that the instruction to rate the stimuli in relation to any oral sensation experienced limited the scope of the scale and its generalizability between individuals and groups. For instance, a strong bitter sensation for a non-taster would not have the intensity experienced by a super-taster (see section 1.4.2), but may be rated the same. To address this limitation, Bartoshuk et al. (2004) stretched the LMS to replace its top anchor with 'strongest imaginable sensation of any kind' in order to provide a universal sensory ruler that could produce taste differences between non-tasters and super-tasters. The new scale was named the general Labelled Magnitude Scale (gLMS). The scale was found to reliably distinguish between taster status groups (Bartoshuk et al., 2004) and has become a widely used measure of taste intensity

(e.g., Bartoshuk et al., 2006; Dinehart et al., 2006; Duffy et al., 2004; Green & George, 2004; Green et al., 2010; Hasse et al., 2009; Hayes & Duffy, 2007; Hayes et al., 2013; Keat, 2008; Lanier et al., 2005; Mattes, 2009; Reed, 2008; Rudenga et al., 2012).

1.4.2. Factors that affect taste intensity perception

The perception of taste intensity is influenced by a number of factors. Firstly, an individual's genetic taster status can greatly affect the magnitude of taste sensations (e.g., Bartoshuk, 2000; Drayna, 2005; Drewnowski, 1997; Tepper, 2008). This genetically inherited trait (Tepper, 1999) defines the ability to taste the bitter compounds 6-n-propythioracil (PROP) and phenylthiocarbamide (PTC). Non-tasters are unable to detect PROP or PTC and are believed to possess two homozygous recessive alleles (tt). Medium-tasters are able to detect concentrations of PROP and PTC and are thought to have one dominant and one recessive allele (Tt). Super-tasters on the other hand, experience the taste of PROP and PTC as stronger than medium-tasters and are believed to possess two dominant alleles (TT; e.g., Bartoshuk, 2000; Prescot & Tepper, 2004). Super-tasters have been shown to perceive a number of tastes more intensely. For example, PROP tasters have reported rating substances such as urea, sucrose octa-acetate, denatonium benzoate (Mela, 1989), sodium, potassium benzoate, potassium chloride (Bartoshuk et al., 1988), quinine (Leach & Noble, 1989), and caffeine (Hall et al., 1975) as more intensely bitter than non-tasters. PROP tasters have also been shown to perceive sucrose (Bartoshuk, 1988) and NaCl (Bartoshuk, 1998) as more intense than non-tasters. Moreover, evidence is emerging that PROP phenotypes show differences in the cortical processing of tastes, with tasters showing increased activity in the dorsolateral prefrontal cortex (dlPFC) and ventrolateral prefontal cortex (vlPFC) (e.g., Bembich et al., 2010).

Relatedly, the density of fungiform papillae located on the anterior portion of the tongue can affect the magnitude of the taste sensation (e.g., Zhang et al., 2008). Fungiform papillae densities have been shown to be different in the three taster groups, being greatest amongst supertasters (Bartoshuk et al., 1994; Essick et al., 2003; Miller & Reedy, 1990b; Shahbake et al., 2005). The density of fungiform papillae or the number of taste buds is directly correlated with the sensitivity of taste

perception (e.g., Zhang et al., 2008) and degeneration or loss of taste perception is accompanied by a decrease in papillae density (Kim et al., 2003; Reed et al., 1999).

Another important factor influencing taste intensity perception is age (e.g., Cowart & Baum, 1985; Methvan et al., 2011; Mojet, Heidema & Christ-Hazelhof, 2003; Murphy & Gilmore, 1989; Weiffenbach, Yamaguchi, Endo & Yoshimura, 2002). In a study investigating detection thresholds in a large (n = 670) sample of 20 – 90 year olds, it was found that salt thresholds significantly increased at 70 + years of age, bitter at 80 + years and sour at 60 + years (Yamaguchi et al., 2002). In a recent meta-analysis of 23 studies (Methvan et al., 2011), 20 articles reported thresholds for sweet, salty, sour, bitter and umami tastes increased with age. On average, the increase in detection thresholds from younger to older cohorts was 11 mM to 21 mM for salt; 0.4 mM to 0.7 mM for sour; 1.4 mM, to 1.8 mM for bitter, 12.4 mM to 16.8 for sweet and 2.5 mM to 5.5 mM for umami.

Lastly, adaptation and habituation are crucial factors that affect the perceived intensity of a stimulus. Adaptation refers to the 'decrement of intensity or sensitivity to a compound under constant stimulation by this compound' (Breslin & Huang, 2006, p. 176). In this sense, adaptation is referring to biological mechanisms such as peripheral and central receptors that become insensitive (or sometimes more sensitive, e.g., Beksy, 1960) as a result of repeated stimulation. Similarly, habituation refers to a response decrement as a function of stimulus repetition, however, it differs from adaptation in that it does not result from receptor adaptation but is a learned mechanism by which we become accustomed to a stimulus over time (Thompson & Spencer, 1966).

Human behavioural studies have shown that repeated stimulation of the same taste stimulus results in decreased ratings of stimulus intensity (Karrer & Bartoshuk, 1991; Gent, 1979; McBurney et al., 1997; Meiselman, 1968; O'Mahoney & Wong, 1989; Overbosch et al., 1991). In terms of adaptation, this effect can arise from a decline in peripheral and central neuronal responses to tastants (e.g., Diamant & Zotterman, 1969; Sato, 1971) and would result in a temporary decline of the perceived intensity of the stimuli. In terms of habituation, such a decline in taste intensity ratings would arise due to decreased attention to the stimuli, and can also result in reduced activations in cortical taste regions (e.g., Wagner et al., 2005).

1.4.3. Taste intensity coding

1.4.3.1. Spatial coding mechanisms

The central coding of taste intensity is considered to occur within insula and opercula regions. Pribram and Bagshaw (1953) found that lesions in the anterior insula (AI) and frontal operculum (FO) resulted in elevated tastes thresholds in macaques. Similarly, in humans, lesions of the ventral insular cortex in a single subject resulted in elevated tastes thresholds for citric acid (Small et al., 1997).

Processing in the AI/FO regions is also critical for supra-threshold taste intensity perception. Intensity response functions generated from taste responsive cells in the monkey AI/FO regions conform to slopes of intensity ratings reported in human psychophysical studies (Smith-Swintosky, Plata-Salaman & Scott, 1991). Moreover, changes in human taste intensity perception occur following AI/FO lesions (e.g., Pritchard et al. 1999; Mak et al. 2005; Simmons et al., 2003). Patients with right hemispheric damage to the insula report decreased intensities to tastes applied to the right side of the tongue, whereas patients with left sided insula damage report decreased intensity to taste applied to the left side of the tongue (Pritchard et al., 1999). Moreover, in healthy subjects, blood-oxygen-level dependent (BOLD) responses to taste stimuli increase as a function of stimulus concentration in the in the AI/FO region (Small et al., 2003).

The amygdala may also play a role in the processing of taste intensity. In rodents, rejection thresholds for bitter tastes are reduced following amygdala lesions (Touzani, Taghzouti & Velley, 1997). Similarly, in humans, surgical resection of the amygdala results in increases in reported taste intensity of bitter substances (Small, Zatorre & Jones-Gotman, 2001a, 2001b). Moreover, in healthy subjects, responses to the intensity of pleasant and unpleasant stimuli have been observed in the amygdala, along with activations in brain stem and primary gustatory regions which respond commensurately with the intensity of the stimuli, independent of its valence (Small et al., 2003).

1.4.3.2. Temporal coding mechanisms

It is generally accepted that temporal neuronal signals play a distinct role in the coding of stimulus intensity (see Lestienne, 2001, for a review). For instance, increased firing rates in primary sensory cortices have been observed for increases in brightness (e.g., Rossi et al., 1996), loudness (e.g., Polley et al., 2004), olfactory

intensity (e.g., Poo & Issacson, 2009) and taste (Scott & Perrotto, 1980; Ganchrow & Erickson, 1970). Moreover, human EEG studies have reported intensity-dependent shifts of the waveform toward shorter latencies and higher amplitudes in ERP deflections for audition (Rapin et al., 1966) and vision (Spekreijse et al., 1973) and olfaction (Hummel & Kobal, 2001) and it is suspected that a similar mechanism operates for taste intensity representation.

Studies investigating EEG and MEG responses to taste of different intensities have found that increases in tastant concentration does result in enhanced amplitudes (e.g., Hummel et al., 2009; Hummel et al., 2010; Kobayakawa et al., 2008; Ohla et al., 2010) and shorter latencies (Hummel et al., 2009; Hummel et al., 2010; Ohla et al., 2010). For example, Hummel et al. (2010) presented participants with weak and strong acetic acid whilst recording EEG. It was found that P1 and N1 latencies were shortened for increased acetic acid concentrations, while P2 amplitudes were increased.

The localisation of intensity dependent gustatory activity was explored in an MEG study. Yamamoto et al. (2003) reported a late gustatory potential at ~350 ms that was enhanced for increased taste intensity. This component was found to originate in both the AI/FO and somatosensory regions. However, the timing of the potential was much later than had been previously reported (e.g., Kobayakawa et al., 1996). It has been suggested that this confound may have arisen from the use of brief transient stimulation (200 ms) which would result in an overlap of ON and OFF responses, affecting the quantification of the ERP (Ohla et al., 2010). In contrast, Ohla et al. (2010) presented participants with electric taste for 1000 ms at both low- [-6 decibels (db)] and high- (34 db) intensities. This study reported ERP deflections beginning as early as 70 ms after stimulus onset. Ohla et al. (2010) also reported intensity dependent amplitude differences, which they were able to source-localise to AI/FO and somatosensory regions.

In a series of MEG studies, Kobayakawa et al. (1996, 1999, 2008, 2012) investigated gustatory evoked magnetic fields in the human PGC. In their 2008 investigation it was reported that the magnitude of PGC activity increased in a concentration dependent manner in response to NaCl stimuli ranging from 30 mM to 1 M, although the perceived stimulus intensity was not recorded. In a later study, Kobayakawa et al. (2012) compared PGC responses to ratings of stimulus intensity and found that increases in PGC activity were congruent with taste intensity ratings.

1.5 Taste Hedonicity

1.5.1. Taste preferences

Multiplexed together with quality and intensity in the response to a tastant is its hedonic value (Hallock & Di Lorenzo, 2006). The hedonic component refers to the extent to which the tastant is liked or disliked and this domain is fundamental in food selection and the control of intake in many species (Breslin & Spector, 2008). Although hedonic responses to foods are somewhat dissociable from the physical parameters of quality and intensity (Breslin & Spector, 2008), we know that the influence of palatability is at least partially dependent on these sensory characteristics (Lundy, 2008). An organism must transform the physical properties of the stimulus into psychological properties of palatability in order to promote ingestion or rejection (Sadacca et al., 2012).

Cross-culturally, food preferences are diverse (Rozin & Vollmecke, 1986), and while early research posited that food preferences support nutritionally adequate diets, in that organisms have innate special appetites for depleted nutrition sources (e.g., Richter, Holt & Barelare, 1938), this has largely been rejected in favour of evidence that food preferences can be learned and environmental factors such as food availability play a critical role in food preference and selection (see Birch, 1999, for a review).

The origins of basic taste preferences, however, are likely utilitarian (Anderson & Sobel, 2003; Cowart, 1981; Desor, Maller & Greene, 1977; Steiner, 1977; Weiffenbach, 1977). For instance, sweetness is common to safely edible foods (e.g., Cowart, 1981; Ramirez, 1990; Steiner, 1977; Steiner et al., 2001; Weifenbach, 1977), whereas bitterness often signifies poison or spoilage (Cowart, 1981; Hladik et al., 2002; Steiner, 1977; Steiner et al., 2001). With very few exceptions (see Nolte et al., 1994 and Lindemann, 1996), non-human animals as well as human neonates show preference and aversion to sweet and bitter stimuli respectively (e.g., Cowart, 1981; Steiner, 1977; Steiner et al., 2001; Weifenbach, 1977).

At birth (and perhaps before) humans posses the ability to detect sweet taste and this system is interacting with mechanisms that control affect (e.g., Harrison et al., 2010; Steiner et al., 2001; Rosenstein & Oster, 1988). For instance, when a sweet solution is placed in the oral cavity, infants relax in the face and sometimes smile

(Steiner et al., 2001; Rosenstein & Oster, 1988). The preference for sweet taste in children is evident worldwide (Desor & Beauchamp, 1987; Mennella, 2008) and remains heightened until early adolescence, before declining in adulthood (Desor & Beauchamp, 1987; Mennella et al., 2011).

Salt taste preferences are thought to develop around 2 – 6 months (Beauchamp, Cowart & Moran, 1986) and are more complex and less understood than sweet taste preferences (Mennella, 2014). Evidence suggest that infants and young adults show greater preference for salt if their mother experienced severe morning sickness (Crystal & Bernstein, 1995; 1998), although the reason for this is unclear. In adults, preference for salt is generally only observed when it is an additive to foods (Huggins et al., 1992; Pangborn & Pecore, 1982), with this cohort displaying an aversion to pure NaCl solutions (Hladik et al., 2002).

Detection and rejection of bitter tastes are present at birth (Desor, Maller & Andrews, 1975; Kajiura, Cowart & Beauchamp, 1992; Steiner et al., 2001). Within hours of birth, infants respond to bitter quinine taste with appropriate facial expressions for the expectoration of the substance (e.g., gape, nose wrinkle, head shake), similar to reactions observed in primates and rats (e.g., Berridge, 1996; Stenier et al., 2001). While strong bitter tastes signify toxins and are highly aversive, mild bitter tastes are contained in numerous human foodstuffs (e.g., cruciferous vegetables, coffee, cheese), which are enjoyed by many (Mayerhof, 2005). The preference for bitter tasting foods is considered to be mediated, in part, by genetic taster status (see section 1.4.2). Those who are non-tasters experience bitter tastes less intensely than medium- or super-tasters and show a greater preference for bitter tasting foods; whereas super-tasters show greater sensitivity and dislike for bitter tasting substances (Bartoshuk, 1989).

Overall, preferences and aversions for basic tastes seem to be innate and develop over the lifespan. As addressed later, however, the hedonic value of basic tastants can be dynamic and modulated by a myriad of non-inherent factors including (but not limited to) culture, appetite and prior experiences (e.g., Bartoshuk, 2000; Berridge, 1991; Fortis-Santiago et al., 2010; Hayes et al., 2010; Pangborn, 1970).

1.5.2. Gustatory hedonic coding

1.5.2.1. Spatial coding mechanisms

Many studies examining the neural representation of hedonic value have highlighted the amygdala as a potential processing site (e.g., Berridge, 2000; Le Doux, 2000; Royet et al., 2001; Zald et al., 1997; Zald et al., 2004). Early studies tended to link this region with the processing of threatening, fearful and aversive events (e.g., Aggleton, 2000; Le Doux, 1986). However, it is now known that the amygdala also plays a role in the processing of positive affect (e.g., Breiter et al., 1996; Cahil & McGaugh, 1990; Schneider et al., 1997).

In terms of taste, it is known that the amygdala receives input both from the primary (Mufson et al., 1981; Norgren, 1976) and secondary (Baylis, Rolls & Baylis, 1995; Carmichael & Price, 1984) gustatory cortices. Evidence of the amygdala's involvement in taste has been reported in animal studies. For instance, in monkeys, amygdala lesions have been shown to change food preferences (Baylis & Gaffan, 1991; Butter, McDonald & Snyder, 1969) and in rats - lesions of the amygdala have been shown to reduce neophobia and taste aversion (Burns et al., 1996; Kiefer & Orr, 1992). Moreover, neurophysiological recordings made in non-human primates (Scott et al., 1993) have shown that some amygdala neurons respond to rewarding tastes (e.g., glucose) and some respond to more aversive tastes (i.e. sour).

In humans, however, observations of hedonic taste responses in the amygdala have been less consistent (Anderson et al., 2003; Mak et al., 2005; Small et al., 2003; Stevenson et al., 2008). Small et al. (2003) manipulated concentrations of quinine sulphate and sucrose such that subjective intensity and hedonic value could be examined independently. fMRI recordings of human subjects showed that the responses in the pons, mid-insula cortex and amygdala corresponded with intensity, irrespective of hedonic value; whereas AI and OFC activations corresponded with hedonic value irrespective of intensity - suggesting that the amygdala processes taste intensity. Furthermore, the right caudolateral OFC was more responsive to the pleasant experience of sucrose, whereas the anterior left OFC was more responsive to the unpleasant experience of quinine (Small et al., 2003). This has been supported by a number of other studies also finding gustatory hedonic responses linked with OFC activations (e.g., Anderson & Phelps, 2002; Davidson & Irwin, 1999; Grabenhorst et al., 2007; Guest et al., 2007; Rolls et al., 1989, 1990, 1996; Rolls et

al, 2006; Yamamoto et al., 2003). Moreover, these results are in line with findings from olfactory research (Anderson et al., 2003) and indicate that there may be spatial segregation of the neural representation of the intensity and hedonics of sensory experience (Anderson & Sobel, 2003; Small et al., 2003).

1.5.2.2. Temporal coding mechanisms

In order to examine the temporal coding of taste hedonics and distinguish this from the coding of taste intensity, Sadacca et al. (2012) presented four concentrations of NaCl (intensity measure), as well as sucrose and quinine (benchmark palatable and unpalatable stimuli) to awake rats while recording neuron responses in the primary gustatory cortex and amygdala. It was found that taste intensity was reflected in the first 500 ms of taste-specific activity in the gustatory cortex, with activations increasing proportionately with rises in concentration. In an overlapping subset of neurons, but several hundred milliseconds later, palatability was characterised by a change in firing rates (Sadacca et al., 2012).

A similar pattern emerged in the central amygdala, but with palatability represented slightly later than in the primary gustatory cortex, suggesting that palatability is initially encoded within the primary gustatory region (Sadacca et al., 2012). Furthermore, the results indicate that while palatability and concentration are processed at distinct times, they are related properties represented by overlapping subsets of neurons. These results highlight the important distinction between processing in primary sensory regions and early sensory processing, in that while palatability was processed by the PGC, it was processed much later than taste intensity. This is consistent with human ERP research which indicates that early processing stages are dedicated to the coding of physical stimulus properties, while later epochs are associated with the processing of higher-order affective or cognitive information (e.g., Hajcak, MacNamara, & Olvet, 2010). These results further suggest that temporal coding mechanisms may provide a key to understanding the coding of gustatory information and specifically, dissociating the information processing of different tastant characteristics.

In humans, stimulus valence has been associated with late EEG processing signals (see Hajcak et al., 2010, for a review). If one stimulus is more emotionally charged than another (i.e., positive versus neutral stimuli) it will show enhanced amplitudes in late (> 300 ms) EEG processing epochs (e.g., Bernat, Bunce & Shevrn,

2001; Krolak-Salmon et al., 2001). When the intensity (or motivational salience) of positive and negative emotional stimuli are matched (i.e., erotic versus fearful), however, equivalent late amplitudes are observed (Schupp, Junghöfer et al., 2004; Schupp, Ohman et al., 2004; Schupp, Cuthbert et al., 2004), making it difficult to dissociate between hedonically positive and negative stimuli of equal intensity. In oscillatory data, however, differences in the processing of positive and negative stimuli and events have been observed. A decrease (ERD) in left-sided alpha-band oscillations has been consistently observed in response to stimuli of positive emotional valence (Balconi & Mazza, 2009, Davidson & Henriques, 2000; Waldstein et al., 2000). Similarly, within theta-band oscillations, right-hemispheric ERS has been observed in response to negative emotional stimuli and left-sided ERS in response to positive emotional stimuli (Aftanas & Golocheikine, 2001).

Within gustatory research, sweet (pleasant) tastes have been associated with a right hemispheric P1 and P300 amplitude increase compared with neutral taste stimuli (Franken et al., 2011). However, as mentioned above, these effects could be attributed to differences stimulus quality or intensity rather than hedonicity. Pleasant tastes have also been associated with left-hemispheric theta- and alpha-band ERD in infants, compared with right –sided theta- and alpha-band ERD for water (Fox & Davidson, 1986). Aversive tastes, like pleasant tastes, have been associated with and enhanced P1 and P300 ERP components compared with water (e.g., Hummel et al., 2010; Hu et al., 2014), again though, these effects could be attributed to the quality or the intensity of the tastant rather than the aversive hedonicity. Unpleasant bitter tastes have been shown to elicit right-lateralised theta band ERD in healthy individuals (Toth et al., 2004). However, the current literature on taste hedonicity contains no EEG studies comparing hedonically positive, negative and neutral taste stimuli, while simultaneously controlling for effects of taste quality or intensity. Thus, no specific conclusions on the EEG processing of pleasant and aversive tastes can be drawn.

1.6. Dissociating the Coding of Taste Quality, Intensity and Hedonicity

When reviewing the literature on taste quality, intensity and hedonic processing, many parallels can be drawn with the regional and temporal coding mechanisms identified for each taste characteristic. For instance, primary gustatory regions

identified in the processing of taste quality (e.g., anterior and mid insula, overlying frontal and parietal operculum: Kobayakawa et al., 1999; Pritchard et al., 1999; Schoenfeld et al., 2004; Small, 2010) have also been shown to be differentially activated for different taste intensities (Kobayakawa et al., 2008; Ohla et al., 2010; Smith-Swintosky, Plata-Salaman & Scott, 1991; Yamamoto et al., 2003). In addition, regions associated with the processing of taste hedonicity (e.g., the amygdala: Baylis & Gaffan, 1991; Butter, McDonald & Snyder, 1969) have been shown to be equally, if not more responsive to increases in taste intensity (e.g., Small et al., 2003). Increased P1 (e.g., Franken et al., 2011; Hummel et al., 2010; Hu et al., 2014; Ohla et al., 2010; Singh et al., 2011), and P300 (Franken et al., 2011; Hummel et al., 2010; Hu et al., 2014; Ohla et al., 2010) ERP components have been observed for differing taste qualities, intensities as well as valences. Although there may be some overlap in the processing of different stimulus characteristics within primary and secondary sensory cortices, other modalities have generally reported functional specialisation within distinct regions and temporal processing patterns (e.g., Engel et al., 1992; Reich, Mechler & Victor, 2001; Zatorre & Belin, 2001; Zeki, 1971, 1978).

One of the reasons that the coding of different gustatory characteristics is so difficult to segregate is the fact that behaviourally, taste characteristics can be very intertwined. Varying taste intensity can have considerable influence on the hedonic value of tastants as well as identification of tastant quality (e.g., Bartoshuk et al., 1978; Dzendolet & Meiselman, 1967; Giovanni & Pangborn, 1983; Mojet, Christ-Hazelhof & Heidema, 2004; Ossebarrd & Smith, 1995; Pfaffmann, 1980). For example, increasing the intensity of a pleasant stimulus may increase its palatability, while increasing the intensity of an unpleasant stimulus can decrease its hedonic value (Pfaffman, 1980). Similarly, high concentrations of a sweet (pleasant) taste can be rated as weak, whereas low concentrations of a bitter (unpleasant) taste can be rated as strong (Pfaffman, 1980, Small et al., 2003). Very often participants are unable to rate these characteristics independently from each other without training (Pfaffmann, 1980; Small et al., 2001b). Despite this, many gustatory processing investigations have examined these features independently, not accounting or controlling for the effects of the other taste characteristics (e.g., Franken et al., 2011; Grabenhorst & Rolls, 2008; Hummel, Genow, & Landis, 2010).

Some studies have sought to control for the intensity or pleasantness of the stimuli by matching tastes based on subjective ratings (e.g., Crouzet et al., 2015; Ohla et al., 2010; Singh et al., 2011). In this way, a single characteristic may be observed by carefully controlling for another. Alternatively, other studies have sought to directly compare the processing of different tastant characteristics in a single study.

Small et al. (2003) administered low and high concentrations of sucrose and quinine sulphate in order that the subjective intensity and hedonicity of the tastes could be measured independently. To do this, subjects visited the laboratory prior to testing and rated the intensity and pleasantness of several concentrations of sweet and bitter solutions. The selected stimuli were yoked independently for each participant so that ratings would fall within the target range. Through such careful manipulation, Small et al. (2003) found that the often-correlated dimensions of intensity and valence are supported by dissociable neural substrates (i.e., the amygdala and OFC, see section 1.5.2.1). These results do not, however, rule out any temporal coding schemes that may be at play in these regions. For instance, findings obtained by Sadacca et al. (2012) using a rat model suggest that both the intensity and hedonicity of tastes are represented in overlapping neurons within the amygdala and primary gustatory cortex, but during distinct epochs.

In the Sadacca et al. (2012) study, the examination of the independent characteristics of hedonicity and intensity was achieved by employing a separate intensity measure (four increasing concentrations of NaCl) and a hedonicity measure (single concentrations of quinine and a concentration of sucrose) in such a way that any neural change resulting from changes in concentration could be attributed to intensity coding and differentiated from patterns emerging from the hedonic processing of quinine and sucrose (Sadacca et al., 2012).

To examine the human temporal dissociation of these characteristics, a similar stimuli set to that of Sadacca et al. (2012) could be employed, with the added benefit of being able to obtain subjective ratings of the tastants to ensure that perceptual differences are present. To our knowledge, no human EEG study has examined the separate coding of taste quality, intensity and hedonicity in this way and with such a diverse stimuli set. This approach could provide a crucial

understanding of the temporal mechanisms involved in distinguishing these characteristics and may clarify the conflicting knowledge gained from fMRI studies.

1.7. Taste and Appetite

1.7.1. Hunger and satiety

Hunger is conceptualised as a physiological cue that fosters feeding behaviours in the event of nutrient shortage (Blundell, Lawton, Cotton, & Macdiarmid, 1996). Identifying hunger also relies on other processes including the availability of the food, previous experiences with the food, and the sensory information the food is providing (Blundell, et al., 2010). Thus, hunger may be said to relate to a combination of physiological and psychological factors, with the physiological messages integrated with the available information from the food source and the environment affecting the resulting behavioural response.

Satiety refers to the state in which further intake between eating episodes is inhibited, thus delaying the onset of the next meal (Blundell et al., 1996; Blundell et al., 2010). Physiologically, satiety is induced after the process of satiation whereby post-ingestive signals generate the release of a number of hormones (e.g., cholecystokinin; CCK, glucagon-like peptide 1; GLP-1 and peptide YY; PYY), which are known to influence satiety (Blundell et al., 2001; Blundell et al., 2010; Halford, Boyland, Blundell, Kirkham, & Harrold, 2010; Halford & Harrold, 2012; Moran, 2000). Satiety can also be affected by sensory food characteristics and cognitive components such as expectancy (Blundell et al., 1987).

Appetite and taste are inherently interconnected, with both systems functioning as means to prompt eating behaviours. The gustatory properties of food play an important role in food selection and consumption (e.g., Blundell et al., 2010) and learned associations made between relevant tastes and their biological consequences encourage the conditioning of taste preferences (see Yeomans, 2000). This association can also be reversed, whereby appetite can directly influence taste perception. Cabanac (1971) found that sweet solutions that are rated as palatable when an individual is hungry become less pleasant after ingestion of glucose syrup; a process he termed '*Alliesthesia*'. Alliesthesia effects have been replicated in many subsequent studies and are considered to derive from post-ingestive signals (e.g., Giza & Scott, 1987, see also Fantino, 1984, for a review). Similarly, the perceived

pleasantness of a food recently eaten to satiety has also been shown to decrease, in a phenomenon known as sensory specific satiety (Hetherington et al., 1989; Rolls et al., 1981; Rolls et al., 1983; Rolls, 1985).

Biological states of hunger and satiety have long been posited to affect sweet taste preference (e.g., Albanese et al., 1955; Mayer -Gross & Walker, 1946; Pangborn, 1959). For instance, Mayer-Gross & Walker (1946) reported that individuals with low blood sugar described greater preference for a high sweet taste than those with high blood sugar levels. Moskowitz et al. (1976) compared participants' intensity and pleasantness ratings for varying concentrations of glucose, NaCl, citric acid and quinine sulphate after fasting and following satiation with a glucose load. The findings indicated that where pleasantness ratings for glucose reached a break point under fasting conditions (ratings initially increased with concentration, reached a maximum point then decreased), no break points were observed in the sated condition; rather the mean pleasantness ratings continued to increase. The authors concluded that following satiation individuals are no longer able to differentiate between the affect and intensity of sweet taste solutions. This study, however, only measured eight subjects in each group so the results cannot be generalised. Furthermore, the ratings for the unpleasant tastes were not reported, so it is unclear whether a diminished discrimination of taste affect and intensity following satiation appears across all tastes, or whether it is specific to palatable or nutrient rich tastants.

However, in a landmark study of 11,456 consumers at a state fair, Pangborn (1959) collected ratings of hunger before asking consumers to taste and rate six samples of canned peaches with varying concentrations of sucrose. It was observed that appetite was unrelated to pleasantness ratings of tastes, a finding that Pangborn (1959) replicated in a laboratory study in sated and hungry participants.

As well as taste preference, appetite may also modulate taste intensity perception (Glokner, Fikentsher & Ulrich, 1986; Kawai et al., 2000; Shigemura et al., 2004; Zverev, 2004). While early studies reported no effect of internal state on the sensitivity to tastes (e.g., Pangborn, 1959), more recent investigations are challenging this view (Glokner, Fikentsher & Ulrich, 1986; Kawai et al., 2000; Shigemura et al., 2004). For instance, Zverev (2004) determined taste thresholds for sucrose, NaCl and quinine sulphate in 16 normal weight males in fasted and sated

states. The findings indicated that sensitivity to NaCl and sucrose was increased (thresholds decreased) during the fasted, compared with the sated state; whereas thresholds for bitter tastes did not change. These results suggest that alterations in taste perception following satiation may only affect palatable or nutrient rich tastes.

In contrast, however, with a larger set of subjects and a more varied tastant stimuli set, Pasquet et al. (2006) reported no differences in taste thresholds between hungry and sated participants. Thus, the current literature on the effects of appetite on taste intensity and pleasantness perception is mixed and it is unclear whether the effects of satiation that were observed for hedonic tone are specific to pleasant and nutrient rich tastes, or can be applied to taste that are more aversive i.e., are aversive tastes equally unpleasant in hungry states?

1.7.2. Appetite and taste processing

The neural substrates of hunger and satiety have been well described (see Ahima & Antwi, 2009, for a review). The hypothalamus and brain stem are major centres for the regulation of food intake (see Oomura & Kita, 1981), and the gut-brain axis modulates appetite and satiety via neuronal and hormonal signals between the digestive system and these brain regions.

The physiological state of hunger has been associated with increased neuronal activity in the hypothalamus and thalamus, as well as increased activations in limbic regions (associated with affect and motivation; e.g., Cardinal et al., 2002), whereas satiety has been shown to activate prefrontal cortical areas associated with response inhibition (Burton et al., 1976; de Graaf & Kok, 2010; Del Parigi et al., 2004; Gautier et al., 2000; Kikuchi et al., 2005; Kobayashi et al., 2004; Simmons et al., 2005; Small et al., 2001; Rolls et al., 1989; Tataranni et al., 1999).

A number of human neuroimaging studies have examined neural responses to chemosensory stimulation under hungry and sated conditions (Del Parigi et al., 2004; Gautier et al., 2000; Gottfried et al., 2003; Hasse et al., 2009; Kringelbach et al., 2003; Tataranni et al., 1999; Uher et al., 1996). When experiencing chemosensory stimuli, neural responses show consistently greater activation within the insula and thalamus under hungry conditions, and increases in OFC activations under sated conditions (Del Paragi et al., 2002; Hasse et al., 2009; Tataranni et al., 1999; Uher et al., 1996). However, the chemosensory stimulation under observation varies between studies; with some examining responses to food stimuli (e.g., Uher et al., 2006) or

liquid meals (e.g., Del Paragi et al., 2002; Kringlebach et al., 2003; Tataranni et al., 1999) and only one to pure tastants (Hasse et al., 2009), so it is unclear whether these studies are observing the same processes (i.e., taste, flavour, reward).

In the Hasse et al. (2009) study, fMRI participants were presented with pure gustatory stimuli including, NaCl, sucrose, citric acid, caffeine and guanosine 5′-monophosphate (GMP) under sated and fasting conditions. It was found that under fasting conditions, all tastes (compared with water) showed greater global brain activations, and this was particularly evident in the insula. Under sated conditions, decreased activations to tastes were observed in limbic regions (involved in emotion and motivation). Overall, sucrose elicited the greatest global activation in both fasting and sated conditions. These results suggest that the state of hunger elicits activations from regions involved in the sensory processing of tastes and satiety deactivates regions associated with motivation and reward, perhaps as a mechanism to terminate food intake. The results also suggests that sweet tastes produced increased activations compared with other tastes during states of both hunger and satiety, highlighting the significance of hedonic factors in appetite and the importance of comparing various stimuli qualities when examining the complex relationship between hunger and taste.

Temporally, changes in ERP amplitude have also been associated with metabolic state. EEG studies have reported decreased P300 amplitudes in hungry compared with sated subjects (Gesiler & Polich, 1990; 1992). However, when viewing food images, ERP amplitudes in hungry participants have been shown to increase (Plihal et al., 2001; Stockburger et al., 2008). Only one study has examined the interaction between taste and hunger using EEG (Jacquin-Piques et al., 2016). In this study, ERPs from frontal and central electrode sites were recorded for seven males and eight females whilst tasting one concentration of sucrose 20 times. EEG was recorded twice and on two occasions. In one session, EEG was recorded three hours after participants consumed their normal breakfast, then again 2.5 hours later (hungry condition). In the other session, the same recordings were taken with the addition of an *ad libitum* lunch between sessions. The authors reported that gERP latencies from frontal electrodes (reported as recording PGC activity) lengthened after lunch, with no changes in amplitude observed.

Given the differences in brain activity observed in hungry and sated participants during fMRI (e.g., Del Parigi et al., 2002; Hasse et al., 2009; Kringlebach et al., 2003; Tataranni et al., 1999), and the reported ERP effects of hunger (e.g., Stockburger et al., 2008; Plihal et al., 2001), the limited findings observed by Jacquin-Piques et al. (2016) is surprising. This may be due to methodological differences, particularly with the manipulation of hunger and satiety. In the previous investigations, hunger and satiety were tightly controlled, largely by testing people after an overnight fast, followed by no food or feeding all participants with a meal constituting half of their required resting state calories (e.g., Del Parigi et al., 2002; Hasse et al., 2009; Tataranni et al., 1999). In contrast, the Jacquin-Piques et al. (2016) study tested people just hours after consuming breakfast, and immediately following an *ad libitum* lunch. Thus, whilst reported hunger differed between the hungry and sated conditions, the differences may not have been great enough to detect changes in neural responses. Moreover, the authors measured responses to only one sweet taste, which may elicit different responses from other tastes (e.g., Hasse et al., 2009). In addition, the authors state that differences emerged from PGC regions, although no source-localisation was performed. Given the differences in the densities of cortical layers, it is impossible to determine the source-localisation of ERP components without employing complex algorithms that account for the inverse problem (e.g., Pascual-Marqui, 2002, see Chapter Two, section 2.5.4). Thus, the authors cannot draw any conclusions as to the mechanisms that they are observing (e.g., taste processing, reward processing, appetite processing) or where these effects originate.

1.8. Taste Expectancies

1.8.1. Expectations

One's experience of the sensory environment can be determined by a collection of the physical features of stimuli, as well as the interaction of these characteristics with the observer's own beliefs, prior knowledge and expectations (Gibson, 1966, 1979; O'Regan & Noe, 2001; Rosch, Varela & Thompson, 1991). This integration of information facilitates the development of a coherent and robust percept (Ernst & Bülthoff, 2004; Gibson, 1966), allowing us to make sense of the world and form predictions of future events and sensory experiences (Gibson, 1966). Given the level

of ambiguity and environmental noise in everyday life, a system that reduces computational burden by incorporating prior knowledge and schemas is an efficient mechanism (Engel, Fries, & Singer, 2001; Taylor et al., 2007).

Summerfield and Egner (2009) define expectations as brain states that reflect 'prior information about what is possible or probable in the forthcoming sensory environment' (p. 403). By experimentally manipulating such prior information (i.e., pre-cueing stimuli), it has been shown that expectations can shorten the perception of time (e.g., Wundt, 1936), lead to faster recognition (e.g., Taylor, Bar, & Fragopanagos, 2007) and increase the accuracy of information processing (e.g., LaBerge, 1995). Moreover, evidence is emerging that expectations can affect the neural processing of sensory stimuli.

The most striking example of this is the placebo effect. It has been observed that expecting analgesic treatment but receiving a placebo (placebo analgesia) results in not only in a reduction of reported pain (see Price, Finniss & Benedetti, 2008, for a review) but also attenuation of activations in pain sensitive brain regions (e.g., Fields, 2004; Petrovic et al., 2005; Wager et al., 2004; Zubieta et al., 2001) and reductions in pain related alpha-band ERD (see Peng et al., 2015). Similar effects have been identified in other sensory modalities (e.g., Bulsing et al., 2010; Todorovic & de Lange, 2012; Todorovic et al., 2011; Wacongne et al., 2011; Wager et al., 2004). For instance, expecting an irritating odour, but receiving a non-irritating odour increases N1 and P3 olfactory ERPs (Bulsing et al., 2010).

Taste perception is markedly susceptible to expectations. An oft cited example of this is the influence colour has on flavour identification (e.g., DuBose, Cardello, & Maller, 1980; Levitan, Zampini, Li, & Spence, 2008; Shankar, Levitan, Prescott, & Spence, 2009; Zampini, Wantling, Phillips, & Spence, 2008). For instance, DuBose et al. (1980) asked participants to identify the flavours of differently coloured fruit drinks. Within this task there were appropriate pairings (i.e., a red cherry drink) and inappropriate pairings (i.e., a red lime drink). The findings indicated that the flavor identification was influenced more by the colouring than the taste, in that green drinks were classified as lemon or lime even when they possessed a cherry flavor and *vice versa*.

Another example of prior expectations interacting with taste perception is the effect that brands and labels can have on assessments of flavour (e.g., Allison & Uhl,

1964; Makens, 1965; McClure et al., 2004; Nevid, 1981; Olson & Dover, 1978; Sheen & Drayton, 1988). For example, Allison and Uhl (1964) gave participants unidentified and labelled bottles of branded beer and asked them to distinguish between them, as well as identify their preferred drink. It was found that, as a group, subjects were not able to identify their preferred brand of beer when it was presented unlabelled. However, when labelled, liking ratings were significantly greater for their preferred brand than for the same beer in a blind tasting test. Similar findings have been reported across many branded products (e.g., Makens, 1965; McClure et al., 2004; Nevid, 1981; Olson & Dover, 1978; Sheen & Drayton, 1988). For instance, McClure et al. (2004) found that participants showed greater preference for Coca-Cola™ when tasted from a cup bearing the brand logo than from an unbranded cup, despite no changes to the physical quality of the cola. Moreover, brand knowledge can have specific effects on perceived sensory qualities. For instance, Makens (1965) found that subjects preferred turkey meat identified as being a well-known brand, to the same meat with no brand identification. When asked specifically about the tenderness of the meat, the participants reported the more tender meat as coming from the branded option, demonstrating that they expected the brand to have superior sensory qualities. Such results demonstrate that sensory distinctions can arise from brand perceptions rather than perceived sensory differences (Allison & Uhl, 1964; Deliza & MacFie, 1996).

Understanding the influence expectations have on taste perception could have significant implications for product development, particularly at a time when there is increasing pressure for industries to reduce the fat, salt and sugar contents of food stuffs (Davidenko et al., 2015; Johansen, Næs, Øyaas, & Hersleth, 2010). Often, healthy food alternatives are associated with negative hedonic expectations (e.g., Bowen et al., 1992; Davidenko et al., 2015; Koster et al., 1987; Light et al., 1992; Wardle & Solomons, 1994). For instance, labelling a novel snack as 'healthy' can result in decreased selection in a canteen (Koster et al., 1987) and labelling products as low-fat can result in decreased ratings of palatability compared with the same product without the low-fat label (e.g., Bowen et al., 1992; Light et al., 1992; Wardle & Solomons, 1994). These phenomena may not be generalizable across all product types (e.g., Aaron, Mela, & Evans, 1994; Johansen et al., 2010; Kähkönen, Tuorila, & Lawless, 1997). However, if we can understand the role expectations play in taste

perception and thus food selection, this may inform measures for developing healthy food alternatives free from the association of negative hedonic expectations (Davidenko et al., 2015).

1.8.2. Models of expectation

Several models based on psychological theories have been developed in order to explain how expectations influence taste perception and food consumption. Assimilation Theory was first proposed to explain how prior expectations could adjust attitudes (Hovland, Harvey & Sherif, 1957). The theory became synonymous with the psychological concept of 'cognitive dissonance' (Carlsmith & Aronson, 1963), which holds that when events are incongruent with an individual's expectations, psychological discomfort arises resulting in a negative hedonic state. To avoid this negative state, people will often assimilate their experience of an event with their expectations. Similarly, Assimilation Theory in relation to taste perception predicts that the perceived acceptability of a tastant will assimilate to the level of prior expectation (Anderson, 1973; Cardello et al., 1994; Cardello & Sawyer, 1992; Oliver, 1977; Olshavsky & Miller, 1972; Schifferstein, Kole, & Mojet, 1999; Tuorila, Cardello, & Lesher, 1994; Van Lange, 1999). More precisely, the model holds that when expectations are high but intrinsic quality is low (negative-disconfirmation), liking will assimilate expectations and result in increased acceptance (Cardello, 2003). Likewise, if expectations are low, but intrinsic quality is high (positive-disconfirmation), liking assimilates expectations and acceptance is decreased.

Although many studies report assimilation effects of expectations on food evaluations (e.g., Makens, 1965; McClure et al., 2004; Nevid, 1981; Olson & Dover, 1978; Sheen & Drayton, 1988), often effects do not conform to this model. For instance, when the difference between expectation and outcome is substantial, pleasantness ratings can be reduced. To highlight this, Zellner, et al. (2001) reported that the expectation that an unusual breath-freshener (Jintan) had a pleasant taste resulted in significantly decreased liking ratings than when the breath freshener was assessed without expectation. In this case, disconfirmation of expectancy led to contrast effects.

The Contrast Theory of expectation (e.g., Dawes, 1972), on the other hand, proposes that when an outcome is less favourable than the expectation (negative-disconfirmation), the contrast between the expected and actual outcome will cause the subject to exaggerate the disparity (Yi, 1990). Few studies support the contrast model as a stand-alone theory of food acceptance. This is because contrast effects have been shown to only occur under certain conditions. For instance, Carlsmith and Aronson (1963) found that when a subject expected either a sweet or a bitter solution, but was presented with the other, the bitter solution was rated as more bitter, whereas the sweet solution was rated as less sweet. In this experiment, therefore, contrast effects accounted for the results from the bitter tastant, whereas assimilation effects accounted for the sweet results.

The Assimilation-Contrast Model (Heider, 1944; Sherif & Hovland, 1961; Wilson & Klaren, 1992) is a hybrid of the assimilation and contrast models. The theory predicts that under low-positive or low-negative disconfirmation (differences between expectation and outcome are small), ratings will follow predictions of the assimilation model. However, in situations of high-negative or high-positive disconfirmation (differences between expectation and outcome are large), the theory holds that contrast effects will occur. Wilson and Klaaren (1992) argue that large discrepancies are difficult to ignore and hence orient attention and promote evaluations. Conversely, minor discrepancies may be undetectable and subjects may simply respond according to their expectations.

With regard to taste perception, The Assimilation-Contrast Model (Heider, 1944; Sherif & Hovland, 1961; Wilson & Klaren, 1992) would predict that liking of a tastant could be enhanced when there are only slight discrepancies in sensory information and by manipulating expectation so that any disconfirmation would be low-positive. This approach provides a plausible explanation as to how disconfirmed expectancies result in assimilation in some contexts and contrast in others. However, there is little evidence specifically relating to taste perception, despite the fact that the model is often cited in reference to this phenomenon (e.g., Deliza & MacFie, 1996).

1.8.3. Expectation and taste processing

A number of key neural processes have been identified for expectation and the prediction of upcoming events in the environment (see Segaert et al., 2013, for a review). In general, evidence suggests that when a stimulus is expected or predicted a suppression of neural activity is observed. This effect can be seen in the processing of faces in the fusiform face area (Summerfield, Trittschuh, Monti, Mesulam, & Egner, 2008; see also, Summerfield, Wyart, Johnen, & de Gardelle, 2011), expected tones in the auditory cortex (Todorovic, Van Ede, Maris, & de Lange, 2011) and predicted emotional stimuli in the limbic cortex (e.g., Ishai, Pessoa, Bikle, & Ungerleider, 2004). Moreover, ACC and OFC activations involved in the anticipation of a variety of emotional stimuli (e.g., Koyama et al., 2005, Nitschke et al., 2006, O'Doherty et al., 2002, Petrovic et al., 2005, Ploghaus et al., 1999, Ploghaus et al., 2003; Wager et al., 2004), may mediate the suppression of responses in sensory cortices (e.g., Petrovic et al., 2005).

Temporally, studies examining the neural underpinnings of expectancy have largely been conducted in language contexts (e.g., Curran et al., 1993; Kutas & Federmeier, 2011; Kutas & Hillyard, 1984; Kutas, Lindamood & Hillyard, 1984; Van Petten 1988; Van Petten & Kutas 1990). In such studies, it has been found that the N400 ERP component (associated with semantic language anomalies; see Kutas & Federmeier, 2011, for a review) is decreased when forthcoming words are expected and increased when the words are less predictable (e.g., Curran et al., 1993; Kutas & Hillyard, 1984; Kutas, Lindamood & Hillyard, 1984; Kutas & Van Petten & Kutas 1990; Van Petten 1988;). The N400 effect has also been associated with congruent and incongruent expectation processing in other modalities, including audition (e.g., Besson & Faita 1995; Painter & Koelsch, 2011), vision (e.g., Bobes, Valdessosa & Olivares, 1994; Proverbio & Riva, 2009) and olfaction (Castle, Toller & Milligan, 2000; Kowalewski & Murphy, 2012), suggesting that this ERP component may represent a generalised mechanism for detecting violations of expectancy.

In terms of taste, no studies have explored the effects of expectancy on EEG data, although a few have explored this relationship in BOLD responses. Nitschke et al. (2006) examined BOLD responses to a bitter taste when participants were either presented with a misleading cue indicating that the taste would be less distasteful than it was (mildly aversive), or a cue with accurate information (strongly aversive).

It was found that the misleading cue led to decreased ratings of aversiveness compared with ratings following the accurate cue. Moreover, PGC activations to the bitter taste were decreased when preceded by a mildly aversive cue rather than by a highly aversive cue. Using the same data, Sarinopoulolos et al. (2006) reported that increased activations in the ACC and OFC - during anticipation of the taste following misleading cues - predicted the decreased activations in the PGC, but only in subjects who reported the greatest discrepancy in their ratings of the tastes following the two different cues.

These studies also examined responses to mildly pleasant and highly pleasant tastes with veridical or misleading pleasantness cues. For these pairings no reliable changes in cortical activations were observed, suggesting that such responses may be specific to the processing of aversive tastes. However, in a later study Woods et al. (2011) found that a 'very sweet' textual cue both enhanced subjective ratings of intensity of a diluted orange juice drink (and to a lesser extent its pleasantness) and increased activations of the PGC. Furthermore, Nitschke et al. (2006) and Sarinopoulolos et al. (2006) did not evaluate taste intensity perception, leaving unresolved whether these fMRI results represent changes in pleasantness or intensity evaluations (Okamoto & Dan, 2013).

In contrast to the previously mentioned studies (Nitschke et al., 2006; Sarinopoulos et al., 2006; Woods et al., 2011), Plassmann et al. (2008) did not find evidence of taste expectancies influencing primary taste processing. In their study participants were presented with pairs of identical wines with different price cues and it was found that pleasantness evaluations were greater when the price cue was higher. This was associated with increased activations in the OFC, but no changes in PGC processes were observed. fMRI data indicated that increases in OFC activations were associated with the increase in liking with higher price, whereas no differences in activations in primary gustatory areas were observed. These data suggest that the expectation effect may be driven by secondary, cognitive processing stages, rather than by changes in primary sensory coding mechanisms.

Overall, while it is clear that top-down expectancies influence the perception of taste, the data pertaining to the neural underpinnings of this mechanism are mixed. This inconsistency is perhaps not surprising given the lack of studies in this area, and the variety of methodologies employed.

1.9. Summary and Thesis Aims

In sum, gustatory perception involves the coding of three fundamental tastant characteristics; quality, intensity and hedonic value (e.g., Bartoshuk et al., 1978; Hallock & Di Lorenzo, 2006; Smith & Scott, 2003; Smith & St John, 1999). Several studies have attempted to uncover how these characteristics are encoded within the brain (e.g., Crouzet et al., 2015; Ohla et al., 2010; Sadacca et al., 2012; Singh et al., 2011; Small et al., 2001b; Small et al., 2003). However, these elements can be somewhat intertwined and there is often difficulty separating the coding of one taste attribute from another (e.g., Small et al., 2001b).

Additionally, taste perception, like other sensory modalities, is not a static phenomenon. Rather, it is dynamic in the sense that it can be moderated by physiological and psychological information (e.g., Bartoshuk, 2000; Berridge, 1991; Fortis-Santiago et al., 2010; Hayes et al., 2010; Pangborn, 1970). The palatability of tastes, in particular, may be greatly influenced by states of hunger and satiety (e.g., Cabanac, 1971). Moreover, top-down information such as prior experience and expectations can play a significant role in flavour preference (e.g., Cardello & MacFie, 2007) as well as shaping ratings of the physical attributes of tastes (e.g., DuBose et al., 1980). Therefore, the gustatory system must determine, distinguish and integrate information from a wide range of sources, both within and outside of primary sensory information, in order to make appropriate ingestive decisions.

Electroencephalography (EEG) has the distinct advantage of high temporal resolution (~ 1 ms), which lends itself well to the study of the earliest stages of information processing and the subsequent transitions from sensory-based perceptual processing to the higher-order cognitive operations that are necessary for everyday functions (Light et al., 2010). Thus, exploration of gustatory EEG would provide critical insights into the temporal processing stages of gustation, a factor thoroughly explored in other modalities. However, because of the temporal stimulus precision required for EEG, complex stimulus delivery techniques are required.

The overarching aim of this thesis was to explore the central processing of taste and the influence of physiological and psychological factors on this mechanism. To achieve this aim required a number of objectives to be met.

The first objective was to devise a taste stimuli set incorporating a variety of tastants that can be combined in a study to measure the separate effects of quality,

intensity and hedonicity. The development of the stimuli set is described in Chapter Two, section 2.3.

The next objective was to develop a computer controlled gustometer device and software that can deliver tastants at a controlled time, volume and flow rate, allowing the simultaneous measurement of behavioural and neural responses to those tastants. This is described in Chapter Two, section 2.4.

Using the taste stimuli set and the gustometer apparatus, we then aimed to explore the separate coding of taste quality, intensity and hedonicity. For this we employed various EEG methodologies, including ERP analysis, ERP source localisation and ERD/S analysis. This is presented in Chapter Three.

With an understanding of the processing of taste characteristics gained from Chapter Three, the next objective was to understand how hunger and satiety affected the processing of pleasant, unpleasant and neutral tastes. To achieve this we measured participants EEG responses (described above) to sweet, bitter and water tastes under hungry and sated conditions. This is reported in Chapter Four.

The next objective was to explore the effect of cognitive factors, specifically expectations, on taste intensity processing. In Chapter Five, we describe how manipulating prior expectations of a tastant can affect not only reported intensity, but also the temporal neural signals associated with intensity coding.

Lastly, we aimed to collate the information gained from our experiments to understand the practical advantages and disadvantages of EEG gustometry in order to inform future studies. This is reported in the General Discussion (Chapter Six).

Chapter Two: Methodology

2.1. Participants

2.1.1. Screening & inclusion criteria

A number of individual differences have been found to influence taste sensitivity as well as food preference and appetite in humans, thus participant characteristics in gustatory EEG studies require careful consideration. Moreover, for health and safety reasons, certain individuals are unable to consume specific substances and such populations need to be omitted from gustatory investigations. Therefore, based on careful review, all participants undertook a screening process prior to the experiments (see Appendix A). The screening and inclusion criteria are described below in terms of their influence on taste sensitivity, taste preference and appetite, and safety.

Taste sensitivity

In all experiments, the participants were required to be between the ages of 19 – 35 as this cohort has been found to possess superior taste sensitivity compared with younger and older age groups (Cowart, 1989; Kaneda et al., 2000; Stevens, 1996). In addition, smokers, and persons suffering from a virus or infection at the time of the study were excluded from participating in all investigations, as these factors are known to reduce taste sensitivity (Pepino & Mennella, 2007; Toth et al., 2004; Vennemann, Hummel & Berger, 2008). We also required that participants were not taking any medications that may interfere with their taste sensitivity. These included antihistamines, chemotherapy agents, antibiotics or anti-depressants (see Douglass & Heckman, 2010).

Prior to the experiments recorded in Chapter Three and Chapter Four, participants' bitter taster status was evaluated (Tepper, 2001). Differences in taster status have been reported to account for a significant proportion of variance in bitter taste sensitivity and may be linked to markers of hedonic value (e.g., Bartoshuk, 2000; Drayna, 2005; Drewnowski, 1997; Tepper, 2008). In Chapter Three, the taster status groups comprised of 22.75 % non-tasters, 54.5 % medium-tasters and 22.75 %

super-tasters. In Chapter Four, the taster status groups comprised of 25 % non-tasters, 50% medium-tasters and 25% super-tasters. Taster status was not recorded in Chapter Five as a result of no significant findings for this factor in Chapters Three and Four.

Taste preference and appetite

Body Mass Index (BMI; weight in kg ÷ height in metres^2) was recorded in all studies. Research has shown that BMI may affect taste perception and preference (Bartoshuk et al., 2006; Drewnowski, 1997; Malcolm et al., 1980; Monneuse et al., 2008; Pasquet et al., 2007; Salbe et al., 2004; Simchen et al., 2006; Tepper & Seldner, 1999), although the evidence for this is mixed (Anderson, 1995; Felsted et al., 2007; Frijters et al., 1982; Grinker, 1978; Malcolm et al., 1980).

BMI has also been shown to affect neural responses to hunger and satiety (e.g., Wang, 2009; Gautier, 2000; DelParigi, 2002). In particular, obese (Karhunen et al., 1997, Gautier et al., 2001, Del Parigi et al., 2002 and Del Parigi et al., 2005) and under-weight (Ellison et al., 1998; Gordon et al., 2001; Uher et al., 2004) individuals account for the greastest variability. Thus, these cohorts (BMI < 18.5; BMI > 30) were excluded from the studies reported in Chapters Four and Five, which examined or controlled for hunger and satiety.

As well as BMI, gender can affect neural responses to hunger and satiety. For instance, satiation from chocolate was shown to produce different brain activations in men and women in areas associated with reward and energy homeostasis (Smeets et al., 2006). Moreover, males show greater neural changes from hunger to satiety than females (Hasse, Green & Murphy, 2011) and females show greater PGC responses to tastes when hungry (Uher et al., 2005). Consequently, only female subjects were recruited for the experiments reported in Chapter's Four and Five, which examined or controlled for hunger and satiety.

Health and safety

For health and safety purposes, individuals with any allergies, intolerances or illness (i.e., diabetes) affected by the ingestion of any of the tastants used in the experiments were excluded from participation.

2.2. Materials

2.2.1. Self report measures

The General Labelled Magnitude Scale

All experiments reported in this thesis employed the general Labelled Magnitude Scale (gLMS: Bartoshuk et al., 2004) to gather subjective ratings of the perceived intensity of various tastants. The gLMS is a semantic scale of perceptual intensity encompassing a vertical line characterised by a quasi-logarithmic spacing of its verbal labels. The vertical scale numerically ranges from 0 – 100, with ticks and semantic labels at the following points; barely detectable (1.4), weak (6), moderate (17), strong (34.7), very strong (52.5), strongest imaginable (100) (see Appendix B).

Instructions

Participants were advised to rate the intensity of tastants by indicating a point on the scale that fits their perception, using the 'strongest imaginable' label (top anchor) to refer to the strongest sensation of any kind (Bartoshuk et al., 2004). Participants were required to decide which term most closely describes the taste's strength and then to refine the rating by placing a mark between that descriptor and the next most appropriate label. For instance, if the participant feels that the sensation is a little stronger than moderate, the mark should be placed on the line in between moderate and strong verbal labels (Simon & Nicolelis, 2002). Participants were also instructed to ensure that the ratios among the numbers reflected the ratios between the sensations (e.g., one sensation twice as intense as another was assigned a number twice as large). Finally, the participant was asked whether they have any questions about how to use the scale. This scale has been found to be relatively straightforward and requires little verbal explanation from the researcher (e.g., Hayes et al, 2013).

The Labelled Affective Magnitude scale

The Labelled Affective Magnitude scale (LAM: Schutz & Cardello, 2001) was employed to measure hedonic ratings of tastants. Like the gLMS, the LAM is a vertical category-ratio scale with verbal anchors quasi-logarithmically spaced. The typical LAM ranges from greatest imaginable dislike which derives a score of -100, to greatest imaginable like, which produces a score of +100. However, because we would often present the gLMS and LAM side-by-side, we altered the scaling of the

LAM to range between – 50 (strongest imaginable dislike) to + 50 (strongest imaginable like) to ensure the scales represented the same 100 point numerical measure, in order that the participants could match their responses (see Appendix C).

Instructions

With this scaling technique, we provided similar instructions as we did with the LMS, in order to maintain consistency. Participants were required to read the verbal phrases dispersed among the scale and to decide which term most closely describes the taste's pleasantness and then to refine the rating by placing a mark between that descriptor and the next most appropriate label.

Appetite scale

All experiments reported in this thesis employed an appetite scale (Flint et al., 2000; Rolls et al., 1999) to assess or control for the effects of hunger and satiety on the processing of tastants. The appetite scale consists of a series of 100 mm visual analogue scales (VAS) in which the participant's subjective sensations are measured. Each question is designed to measure slightly different subjective appetite sensations, which all encompass the motivation to eat and relate to subsequent food intake (Alison & Baskin, 2009; Flint et al., 2000; Rolls et al., 1999). These included hunger, fullness, desire to eat, prospective consumption, thirst and nausea (see Appendix D). The questions are designed to reflect the intensity of that particular state at that time, typically before or after the consumption of a meal.

Instructions

The appetite scale consists of seven 100 mm VAS, which are preceded by a question (e.g., 'how hungry do you feel at this moment') and the subject is instructed to place a mark on the VAS indicating how they feel at the time the measurement is taken. Each line incorporates opposing verbal descriptors at each end (i.e. from 'not at all' to 'extremely'), and participants are asked to consider these as extreme labels (i.e., the least and most hungry they have ever felt). A score is derived by measuring the distance in mm from the end of the left line to the mark placed by the participant.

2.3. Taste Stimuli

Rationale

In order to select an appropriate range of taste stimuli for the EEG studies, we conducted an extensive preliminary series of taste tests, assessing ratings of the quality, intensity and pleasantness of different concentrations of salt, sweet and bitter solutions. The criteria for selection for the experiment reported in Chapter Three were that the tastes could be formed into clusters that were easily recognised and differentiated for their taste qualities (salt, sweet, bitter); showed significantly different levels of rated intensity (weak, medium, strong) and differed in hedonic value (pleasant, unpleasant). For the study reported in Chapter Four, we required a pleasant and unpleasant taste that were of equal intensity but dichotomous in pleasantness. For the experiment reported in Chapter Five, we required two pleasant stimuli that differed in intensity.

Methodology

In this preliminary study, a panel of 50 screened (see section 2.1) participants aged 19–35 years (M = 22.22, SD = 3.17, 25 females) rated 81 x 1 ml tastants for quality, using a nominal scale (salt, sweet, bitter), intensity (gLMS; Bartoshuk et al., 2004) and pleasantness (LAM; Schutz & Cardello, 2001). The tastants were nine concentrations each of: quinine hydrochloride (QHCl; 0.000001 M, 0.000003 M, 0.00001M, 0.00003 M, 0.0001 M, 0.0003 M, 0.001 M, 0.003 M, 0.01 M); NaCl (0.01 M, 0.03 M, 0.05 M, 0.1 M, 0.3 M, 0.5 M, 1 M, 3 M, 5 M), and sucrose (0.005 M, 0.01 M, 0.03 M, 0.05 M, 0.1 M, 0.3 M, 0.5 M, 1 M, 3 M). All stimuli were presented at room temperature (23 °C) and were administered from Eppendorf tubes. Taster status groups comprised of 38% non-tasters, 42% medium-tasters and 20% super-tasters.

Instructions and rating scales were presented on a Cathode Ray Tube (CRT) monitor using Psychopy 2.1 (Peirce, 2007). The initial screen requested participants to ‘taste sample number X’ informing which numbered sample to taste. Participants were asked to taste the solution for 3 – 5 seconds, before pressing space. The next screen required the participant to select whether the solution was ‘sweet’, ‘salty’ or ‘bitter’. Following this, they rated the intensity and pleasantness of the taste using the gLMS and LAM described above. Lastly the participants were required to rinse using a bottle of distilled water until they could no longer taste the solution and spit into a spittoon, before pressing space to begin the next trial. The paradigm was designed to allow at least 20 seconds between stimulus presentations and each taste

was repeated three times. Participants who scored < 75 % on the taste quality recognition measure were removed from analysis (n = 9).

All tastants were prepared under sterile conditions using freshly distilled water and transferred from glass bottles to 1 ml food-grade Eppendorfs using a pipette. Tastants were refrigerated for no longer than three days before being disposed of if unused. Eppendorfs were stored at room temperature (set at 23 °C) overnight before testing days. All 81, 1 ml samples were individually prepared and labelled for each of the 50 participants (4050 total). Each experiment took approximately 1 hour to complete. This part of the thesis took around 4 months to complete and took place during the development of the gustometer.

Results

To investigate taste quality recognition, the mean percentage of trials in which participants incorrectly identified the tastes was calculated. Taste concentrations that were incorrectly identified more than 33 % of the time were deemed inappropriate for further testing (Figure 2.1), as this indicated that these tastes may not provide a strong enough percept to invoke neural responses that could be detected using EEG. The excluded tastes as a result of this analysis were QHCl (bitter) samples 0.000001 M, 0.000005 M, 0.00001 M; NaCl (salt) samples 0.01 M, 0.03 M and sucrose (sweet) samples 0.005 M, 0.01 M, 0. 03 M.

To investigate taste intensity and hedonicity, the mean intensity and pleasantness ratings for each taste and each subject were calculated (Figure 2.2). A series of within-subjects ANOVAs examining the effects of concentration on intensity and pleasantness ratings were conducted. Significant main effects of concentration on intensity ratings of bitter, F (8, 136) = 38.63, p <. 001, ES =. 69; salt, F (8, 160) = 97.92, p < .001, ES = .83, and sweet samples, F (8, 160) = 9.53, p = .002, ES = .91, were observed whereby intensity ratings increased with concentration. A significant effect of pleasantness was also observed for bitter, F (3.68, 80.25) = 22.48, p <. 001, ES = .55, and salt samples, F (8, 144) = 22.48, p < .001, ES = .62, with pleasantness ratings decreasing with concentration. There were no effects of pleasantness for sweet tastes (p = .62). Paired sampled t-tests (Bonferonni corrected) were conducted to determine significant pleasantness and intensity differences between individual samples.

As a result of this analysis, five tastes were selected for the studies reported in Chapter Three, which examined taste quality, intensity and hedonic processing. These tastes were categorised into quality conditions of bitter, salt and sweet; intensity conditions of weak, medium, and strong and hedonic conditions of pleasant and unpleasant. The tastes included three concentrations of a salty taste (0.05 M, 0.1 M and 0.3 M NaCl), one bitter taste (0.0003 M QHCl) and one sweet taste (0.3 M sucrose). Figure 2.2 shows the mean intensity and pleasantness ratings for each taste.

For the intensity manipulation, we selected these three concentrations of NaCl because they showed differences in rated intensity [weak, medium, strong; *t*s (40) > 10.41, *p*s < .002], but no differences in pleasantness ratings (*p*s >. 626). In addition, we matched the high concentrations of QHCl and sucrose to the highest NaCl (*p*s > .202) to complete the 'strong' condition. For pleasantness, we clustered the salt and bitter solutions (each rated as unpleasant) to form an 'unpleasant' condition which differed significantly in hedonicity ratings from the 'pleasant' (sucrose) condition, *t*s (40) > 30.10, *p*s < .001. For the EEG study we added a water condition, which acted as a neutral intensity and pleasantness measure.

In Chapter Four we examined the effects of hunger and satiety on the processing of pleasant and unpleasant tastes. We selected the same bitter and sweet tastes that we selected for Chapter Three (0.0003 M QHCl, 0.3 M sucrose), for the reason that they differed in pleasantness but not intensity ratings, and were easily distinguishable for their taste qualities. We also added water as a neutral condition.

For Chapter Five, which examined the influence of expectancy on the processing sweet tastes, we utilised a weak (0.05 M) and a stronger (0.3 M) sucrose concentration for their significantly different intensity ratings, t (39) = 6.86, *p*s < .001, but not pleasantness ratings (p = .09).

To examine whether individual differences affected the quality recognition, intensity ratings and pleasantness ratings of the selected tastes, we conducted series of mixed ANOVAs. There were no effects of gender, BMI, and taster-status on taste quality recognition (*p*s > .178), intensity ratings (*p*s > .121) and pleasantness ratings (*p*s > .244) for any of the tastes selected for the experiments. However, in order to further ensure individual differences did not influence experimental results, we included a taste quality recognition test in our screening procedure for each study, to ensure the participants recognised the tastes used and also recorded or controlled for gender, BMI and taster-status.

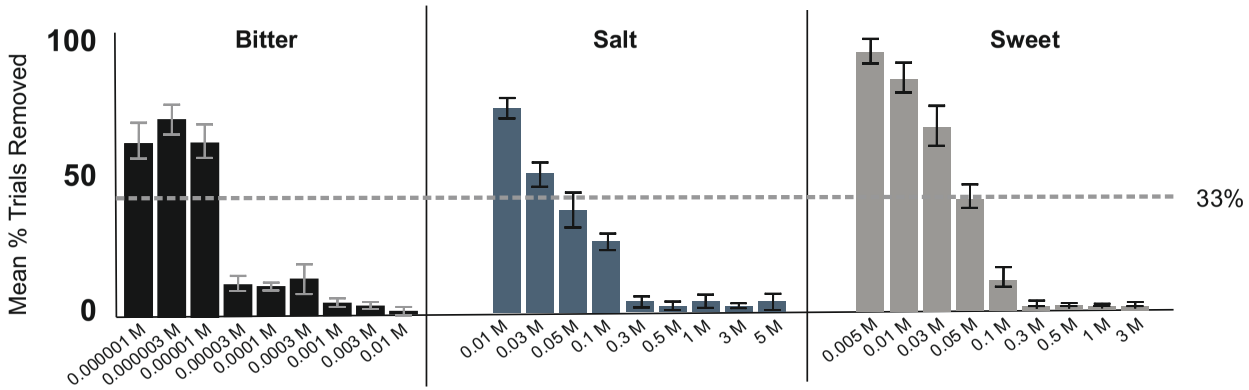

Fig. 2.1. Bar charts showing the mean % correct taste quality recognition for each taste concentration and a dashed line indicating the cut off point for inclusion in the studies

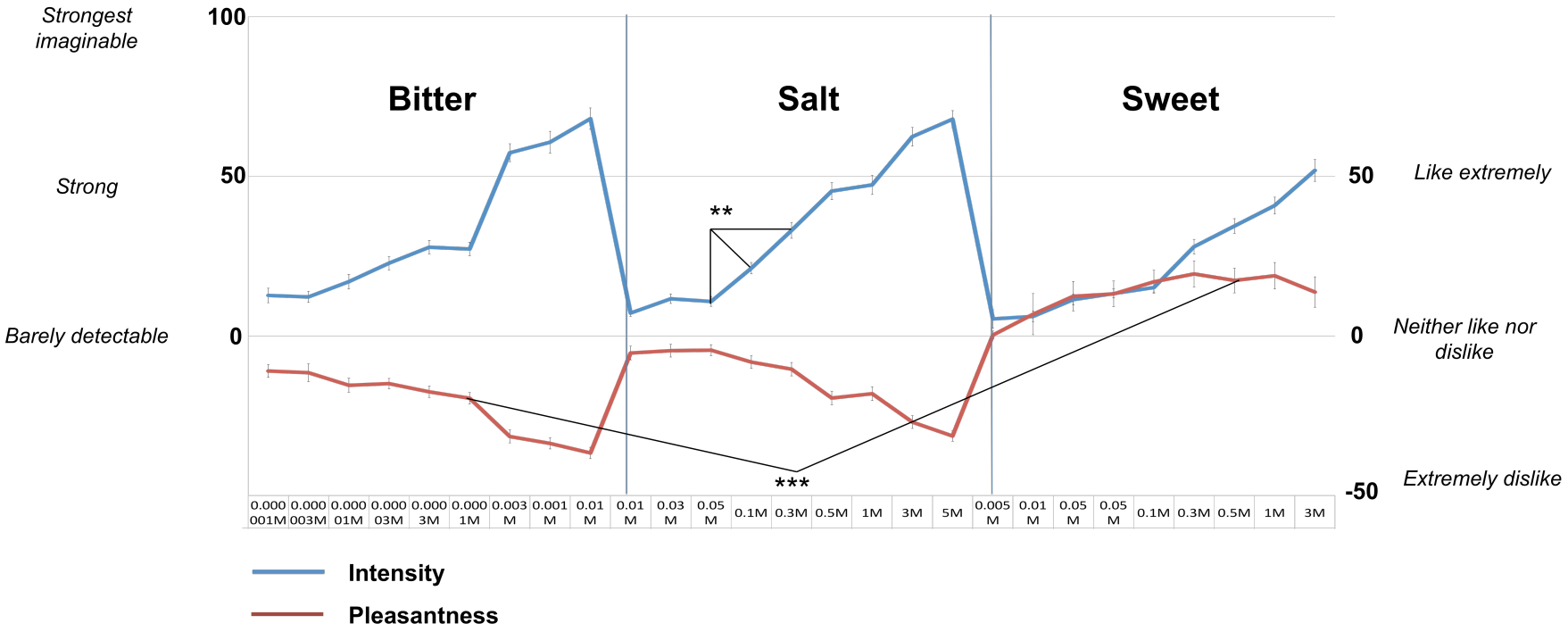

Fig. 2.2. Line graphs indicating the mean intensity and pleasantness ratings for all taste concentrations, with bars representing standard error. Significant differences in intensity and pleasantness ratings have been highlighted. Astericks indicate significance level: ** $p < .01$, *** $p < .001$.

2.4.The Gustometer

2.4.1. Background

In order to examine gERPs, a gustometer mechanism is required to present taste stimuli with steep rise and fall times and at millisecond temporal precision, in order to achieve a good summation of the evoked potentials over trials (Ohla, 2012). Moreover, gustatory EEG studies require many stimulus repeats (30 – 40 per condition/tastant; Mizoguchi et al., 2002) in order to observe neural responses and the tastant must provide a strong enough percept (high enough concentration) in order to evoke neural changes. This means that rinses between each tastant are required along with long ISIs in order to avoid habituation and adaptation (Mizoguchi et al., 2002). Thus, these studies require sophisticated equipment and taxing experimental conditions. Until recently (e.g., Kobayakawa et al., 1996, 1999) such mechanisms were unavailable, meaning that very few gustatory EEG studies have emerged and the gERP is largely uncharacterised.

A number of different gustometer mechanisms have been developed, each with distinct advantages and disadvantages. For instance, the electrogustometer (e.g., Kirchner et al., 2004; Mushimoto et al., 2005; Ohla et al., 2009; Ohla et al., 2010; Saito et al., 2001; Sato & Kamata, 1983) delivers square wave pulses and has the advantage of providing temporal precision in stimulus onset (Yamamoto et al., 2003). Moreover, because no liquid solutions are used, no rinsing is required leading to shorter testing durations than those involved in liquid gustometry (Yamamoto et al., 2003). However, with electrogustometry only responses to sour or metallic taste qualities are registered, so it cannot be used to determine taste quality or hedonic-specific perception (Stillman et al., 2003). As such, this method provides a simulation of taste perception that may bear little resemblance to real life taste experiences.

An alternative method for gustometry involves a mechanism that flows liquid tastants on the tongue within a stream of taste-free liquid, often with the insertion of air-bubbles to prevent mixing between tastes (e.g., Crouzet et al., 2015; Kobayakawa et al., 1996, 1999; Iannilli et al., 2012; Onoda et al., 2005; Singh et al., 2011; Singh et al., 2015). Like electrogustometry, these devices allow for precision timing in

stimulus onset and controlled trial-to-trial variability in the tastants delivered (Toepel & Murray, 2015). Moreover, the continuous spraying of the tongue theoretically habituates the surface to somatosensory stimulation (Kobayakawa et al. 1996b; Singh et al. 2011), thus reducing the likelihood of an overlap of somatosensory and gustatory neural responses. However, a constant flow of liquid invariably results in frequent swallowing motions, particularly in studies in which the participant is advised to swallow freely. This results in masseter muscle movements that can generate substantial artefacts within EEG data (Iannilli et al., 2012). Moreover, presenting so many stimulus repeats within a constant flow of liquid can be lengthy and taxing for participants who have to endure long wash-out periods between trials and maintain open mouths and an immobilised tongue for the duration of the study (Toepel & Murray, 2015). This approach may also result in participant discomfort due to the volume of liquid ingested.

2.4.2. Our gustometer

Taking all of the aforementioned challenges into account, along with resources that were available to us, we established a gustometer system that would spray 1 ml liquid tastants on to the surface of the tongue at a flow rate of 30 ml/min and a rise time of less than 0.02 s that would be followed by a short, 2 ml distilled water rinse. We created a program that allowed for the remote control of the system, precise stimulus onset timing and manual initiation of individual trials. We also included masseter electromygraphy (EMG) along with video monitoring to ensure all trials were initiated in the absence of swallowing motions. These measures did not discount somatosensory potentials, but did create a more realistic taste experience. Experimental procedures were designed to allow for an ISI of at least 25 s. There was no continuous flow of liquid that would results in participant discomfort and frequent swallowing motions.

Mechanism

The gustometer was constructed from eight identical diaphragmatic pumps (KNF Stepdos FEM03.18RC, KNF Verder, Vleuten, The Netherlands; Bult et al., 2007) sourced from Unilever (R & D, Vlardingen, The Netherlands), housed in a custom built frame allowing for tubing to be connected to bottles sitting below the pumps. The pumps were connected via serial port to a Black Box interface (Terminal

Eliminator Plus, Black Box, Lawrence, PA) housed on a separate shelf of the frame. The Black Box allowed communication between the pumps and a computer running custom built programs using psychopy open-source software (Pierce, 2007). Teflon tubing (1.6 mm internal diameter) was used to transport liquids from bottles to the pumps and from the pumps to the participants. Teflon tubing from 6 pumps (no more than six were utilised in any study) was passed thorough an opening in the wall in the EEG chamber to an 8-channel input: 1-channel output manifold (Inacom, Veenendaal, The Netherlands), attached to an adjustable-height retort clamp stand (RVFM Laboratory Retort Stand Set, Rapid Electronics, UK) positioned near the participant. The manifold contained one-way check valves in order to prevent mixing of the liquids. A 10 cm disposable saliva ejector (Topdental, UK) was placed around a 10 cm length Teflon output tube from of the manifold, which delivered the tastes to the participants. The saliva ejector was changed for each participant to prevent contamination. Figure 2.3 shows a schematic diagram of the gustometer and its interaction with the EEG system and participant.

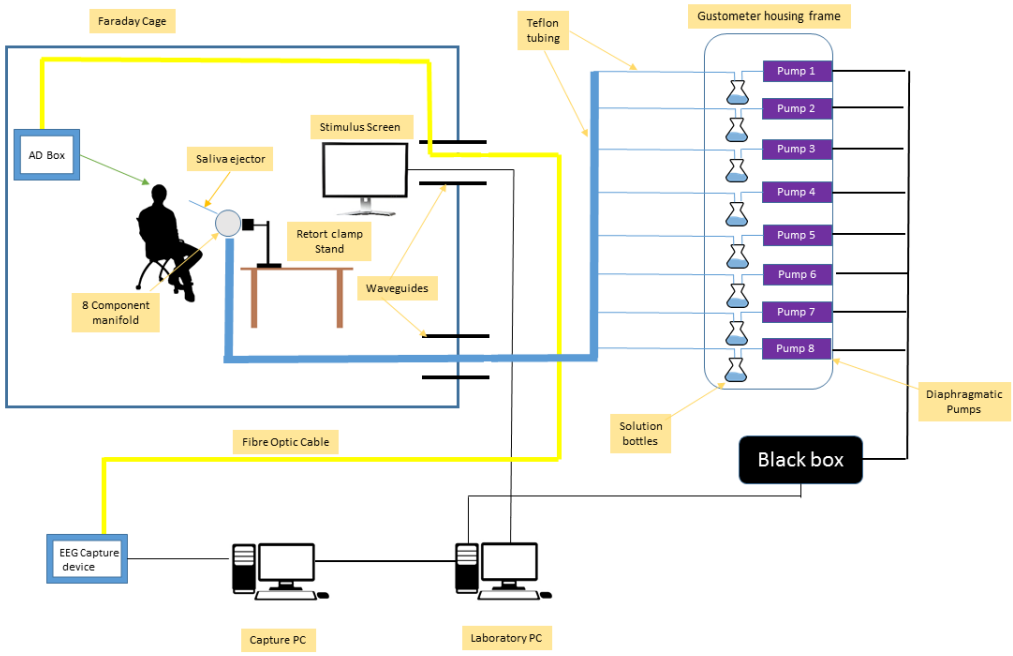

Fig. 2.3. A schematic diagram of the gustometer and it's interactions with the EEG system and the participant.

Electrical grounding – discharging static

When liquid is passed through thin tubing the electrostatic charge of the flowing matter generates a build up of static electricity in a process known as flow electrification (see Dhogal, 1986). When the tubing is plastic or Teflon material, and the liquid is spraying, the charge level is greater. As well as being a safety concern, static electricity can greatly interfere with EEG data. To overcome this severe limitation, we employed a grounding mechanism that created a connection between the source of the charge and the ground, so that any accumulated charge could be discharged. A copper wire was inserted into an unused manifold channel and attached by a length of insulated copper wire to the Faraday cage. Thorough testing conducted by the University of Liverpool's electrical team ascertained that the build up of static from the flowing liquids had been effectively discharged and the gustometer was safe for use.

Water sensor – rise time

In order to calculate the rise time from each pump we used a water sensor device (VELLEMAN KIT, NV, Belgium, 4X). The device traditionally uses resistors, a transistor and a speaker. It works on the principle that water is conductive so when it reaches the contacts it completes a circuit and sets off the speaker. In this case, the circuit was modified so that instead of a speaker output, a signal was transmitted to a computer so that the time difference from the onset of the pumps to the water reaching the sensor could be calculated. We conducted 30 trials for each pump and it was found that the time taken from the return of the pump signal, (pump communicating to the computer that it had received the signal to start), to when the water reached the sensor was always within 0.02 s for all pumps (mean rise-time = 0.004 s, SD = 0.008 s, no significant differences between pumps, ps > .083), providing that they had been primed with fluid. Therefore, the experimental program was designed so that the return signal from the pump also acted as a taste onset trigger for the EEG data. The water sensor device is 45 mm x 70 mm in size so the measurement of online rise-times (intra-orally) was not possible and thus had to be computed separately. Future designs could look to condense such a sensor into a compact mechanism that can be placed intra-orally for a more accuate, online assessment of rise-time. A schematic diagram of the water sensor is presented in Figure 2.4.

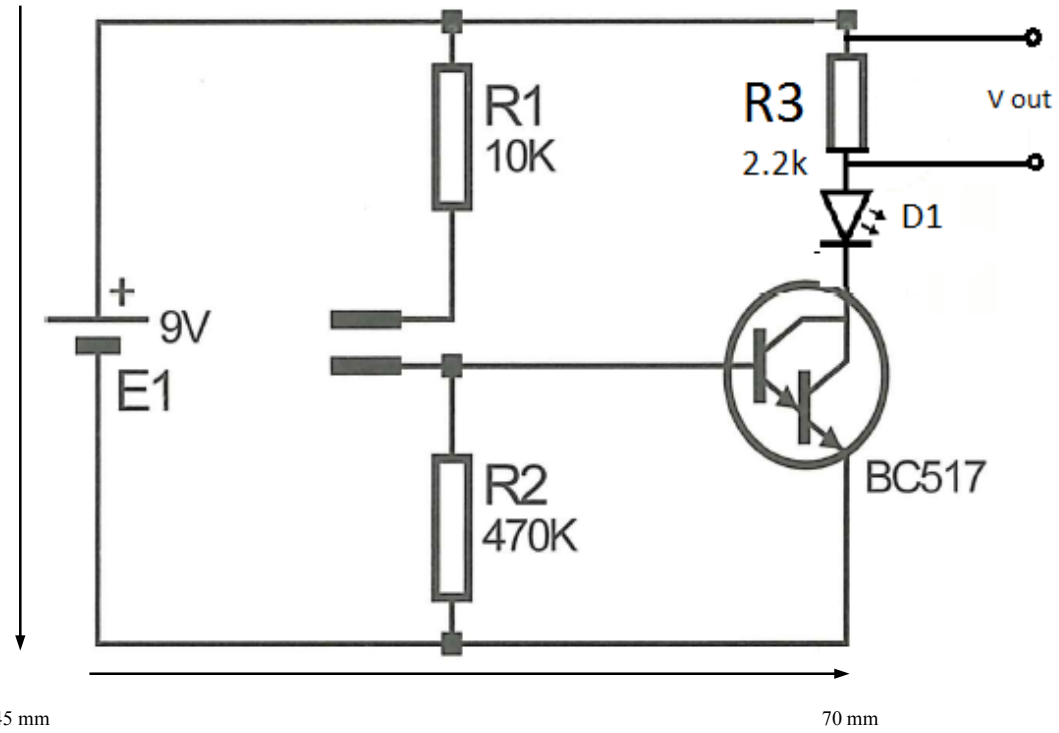

Fig. 2.4. A diagram of the water sensor (R = Resistor, E1 = 9 volt battery, BC = Transistor, D = Light Emitting Diode, v out = voltage output; VELLEMAN KIT, NV, Belgium)

Program

We custom built a program within Psychopy (Peirce, 2007) that allowed for the remote control of the gustometer system (see Appendix E for an example of the program script). The software communicates to the pumps via serial port and indicates which pump should be activated and at what flow rate and duration. The onset of each taste stimuli was communicated from the software via a parallel port trigger to the EEG acquisition system when the computer received a returning signal from the selected pump.

To ensure the onset of the tastant was not concomitant with muscle movements associated with swallowing, we added a manual trial initiation feature to the program sequence. This ensured that a trial could only be initiated when the experimenter was certain that no unwanted movements were being made. This was achieved by the researcher monitoring EMG and video footage of the participant and pressing 'space' when the taste onset was appropriate.

Unfortunately, the KNF Stepdos pumps (KNF Verder, Vleuten, The Netherlands) sometimes do not respond to initiation signals, or shut down entirely during the course of the experiment. While this is generally solved by restarting the pumps, it does create problems within the data. The EEG system would still receive a trigger indicating the condition of the trial that was meant to have occurred. Therefore, in order to avoid including trials in the analysis where the pump failed to pump, we added a trial check procedure into the program. This involved a period of several seconds after the taste onset where the experimenter viewed a small dot on the screen, which prompted them to enter a response of whether the trial was good (g) or bad (b). When a bad trial was indicated it created a trigger within the EEG data (255) that meant that when cleaning the data offline, the experimenter could discard all trials that preceded a 255 event. The triggers were also included in the Psychopy output file so that behavioral data could be filtered to only include good (g) trials. Moreover, when a bad trial was indicated, the rest of trial sequence was aborted and a new trial was started.

Health & Safety

After each testing session, all pumps were re-primed for 60 seconds, saliva ejectors were disposed of and replaced, and the output tubing was sanitised. After each

testing day, the system was cleaned. Firstly, the system was rinsed for 10 minutes using distilled water. This was achieved by placing the pump input tubes into bottles of distilled water and setting the pumps to run for ten minutes at a flow rate of 30ml/min. Following this, the tubing was sterilised by pumping diluted (0.6% V/V) sterilising fluid (Milton BabyCare, Newmarket, UK) through each tube and leaving for 15 minutes before rinsing again with distilled water for 10 minutes.

Summary

The development of this system was a complex process that took almost two years to complete. This involved a substantial research and design process, sourcing of novel parts, mechanical set up, custom software design, electrical grounding, rise-time calculations and extensive testing (described above). Figure 2.5 depicts the timeline of the gustometer development process and the people involved in each development stage.

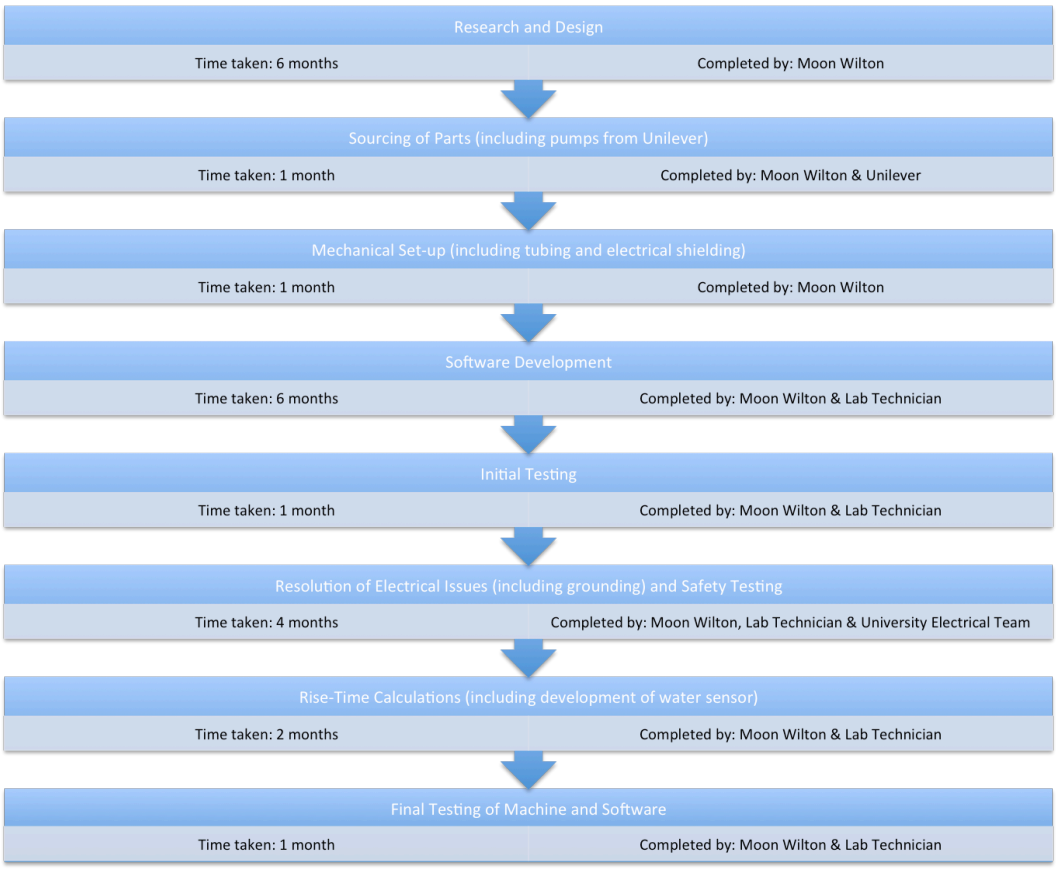

Fig. 2.5. A timeline of the gustometer development process (22 months total) including the time taken to complete each stage and the people who assisted.

2.5. EEG Methodology

2.5.1. Physiological basis for the EEG signal

Electroencephalography refers to the measurement, amplification and recording of minute electrical signals originating from neurons within the cerebral cortex. Within the brain, action potentials transverse long axonal distances to the nerve terminal where neurotransmitters are released. This induces excitatory and inhibitory postsynaptic potentials (EPSP and IPSP). The duration of action potentials is too short (< 2 μs) to contribute to scalp-recorded EEG (except during synchronous events), whereas postsynaptic potentials are much longer (> 100 ms). Therefore, both EPSP and IPSP represent the most significant source of EEG signals. In particular, the electrical activity detected by EEG can be attributed to the summation of electrical field potentials generated by many simultaneous and nearby EPSPs or IPSPs, usually within vertically oriented pyramidal neurons (Bucci & Galderisi, 2011; Goff, Allison & Vaughan, 1978; Goldstein, 2009; Luck, 2005; Shepherd, 1974).

2.5.2. Time-domain activity: Event-related potentials

ERPs refer to EEG voltage changes that are time-locked to the onset of an event (Lopes da Silva, 2005). As such, ERPs are useful for quantifying neurophysiological differences in responses to different groups and conditions (Duncan et al, 2009). One of the most prominent advantages of ERP analysis is that they provide covert, online measurements of stimulus processing making it possible to determine which temporal stage of processing is affected by a specific experimental manipulation (Luck, 2005).

ERPs are elicited by a wide range of sensory, cognitive or motor events and can be divided into two major categories; early and late components. Early components, or waves, occur around the first 100 ms after stimulus onset and are deemed ‘sensory’ or ‘exogenous’ and depend largely on the physical properties of the stimulus. Late components, on the other hand, are deemed ‘cognitive’ or ‘endogenous’ ERPs and reflect more evaluative forms of information processing (Sur & Sinha, 2009).

ERP waveforms are typically described in terms of their latency and polarity of amplitude. As mentioned in Chapter One (section 1.2.3), a P1, or P100 wave refers to the first positive amplitude defection and occurs roughly 100 ms after stimulus onset. Functionally, this component is usually interpreted as an indicator of preferential selection of sensory input (or the suppression of unattended information) and has been shown to be involved in the processing of low-level features of auditory (Waldo et al., 1992), visual (Luck, 1995) and somatosensory (Fukushima et al., 1976) stimuli. The N1 or N100 component is generally assumed to reflect selective attention to low-level stimulus characteristics and discrimination processing (Hillyard et al, 1973; Vogel & Luck, 2000). P2 is the second positive amplitude deflection occurring after 200 ms and, unlike P1 and N1; P2 is associated more with cognitive, rather than sensory processing. The P2 component has been identified during cognitive tasks involving feature detection (Luck & Hillyard, 1994), selective attention (Hillyard et al., 1973) and memory (Golob & Star, 2000). N2 is a negative deflection around 200 ms and is considered to be task dependent (e.g., Johnson, 1989) and stimulus dependent (Allison et al. 1999). N2 differences are most commonly reported in cognitive paradigms such as the Go/No-Go task (e.g., Eimer, 1993) and mismatch negativity (MMN; Naatanen et al., 1993) and are thought to be associated with response inhibition. N400 is a negative amplitude deflection around 400 ms after stimulus onset. This ERP component is associated with unexpected outcomes (particularly semantic language anomalies, see Kutas & Federmeier, 2011, for a review). Lastly, the P300 and late positive potentials (LPP) are positive amplitude shifts occurring on or after 300 ms post-stimulus onset. The P300 and LPP are the most extensively researched ERP components. Functionally, these late components have been shown to represent a diverse range of higher-order cognitive roles, including attention (Overtoom et al., 1998), memory (Donchin & Coles, 1988), emotion (Hajcak et al., 2010; Holt et al., 2009), arousal (Cuthbert, 2000) and top-down control (Johnson et al., 1986). Figure 2.6 (a) illustrates typical ERP waveforms and the functionally distinct ERP components.

2.5.3. Gustatory ERPs

Gustatory ERPs are derived from the EEG after oral chemical or electrical stimulation. Until relatively recently (Kobal & Plattig, 1978; Kobal., 1985), the use

of electrophysiological techniques for the functional exploration of the gustation was limited mainly due to the lack of adequate stimulus delivery techniques to produce controlled and transient chemosensory stimulus (Moncrief, 1962). Chemosensory ERPs, as a whole, usually exhibit a very low signal-to-noise ratio (Boesveldt et al., 2007; Lotsch & Hummel, 2006; Rombaux et al., 2007). For example, Lotsch and Hummel (2007) were unable to identify reproducible olfactory ERPs in approximately 30% of subjects. Unlike visual or auditory stimuli, the presentation of chemosensory stimuli is susceptible to temporal jitters, affecting the ability to time-lock neural responses to the stimulation. The existence of temporal jitters implies that the EEG responses are no longer stationary across trials meaning that responses would be distorted, or cancelled out during averaging procedures (Huart et al., 2012). Moreover, ERPs by nature are small voltage changes and, as such, a large number of trials are required in order to adequately measure them. This is particularly the case for gustatory ERPs, which have been shown to require extensive repeated stimulation in order to detect fully articulated components (Mizoguchi et al., 2002). As a result, gustatory ERP components remain the most elusive of sensory EEG signals.

2.5.4. Source localisation of ERPs

As described in section 2.5.1, EEG signals are generated by current flows that are associated with the transmission of information between populations of neurons. The objective of source localisation is to determine the spatial location of these populations by modelling a number of current dipoles belonging to a source space. The localisation of EEG activity is difficult to precisely determine. From the origin to the source electrodes, the signal must pass through cerebrospinal fluid, dura, skull and scalp, each of which has different conductive properties (Northrop, 2012). Moreover, this activity propagates in all directions by volume conduction and crosses only good conductive layers. For instance, it does not cross the inner borders of the skull - rather it crosses in the layers of the dura and is detected from the skull capacitive charge distribution that occurs in the layers (Northrop, 2012). Thus, the relationship between electromagenetic field activity detected by EEG and current source is not one-to-one. This creates a challenge known as the ‘inverse problem’ (Grech et al., 2008).

In the generic form of the inverse problem there are $^{N}_{E}$ instantaneous extracranial measures and $^{N}_{V}$ voxels in the brain (determined by uniformly subdividing the solution space), with $^{N}_{V} >> {}^{N}_{E}$. At each voxel there is a point source that may be a vector with three unknown components (i.e., the three dipole moments) or a scalar (unknown dipole amplitude, known dipole orientation). A solution to the inverse problem involves the computation of images of electrical neuronal activity based on extracranial measures (Pascual-Marqui, 2002). However, the inverse problem is ill-posed since the solution is not unique (since $^{N}_{V} >> {}^{N}_{E}$) or stable (is highly sensitive to small changes in noisy data) and there are many more unknowns than equations to be solved. Thus source localisation requires complex mathematical equations and estimations.

A number of methods have been developed to solve the inverse problem in order to effectively source localise EEG components. These include parametric methods such as Brain Electrical Source Analysis (BESA; Baillet, 1998), Beamforming (Baillet, Mosher & Leahy, 2001) and the Multiple-Signal Classification algorithm (MUSIC; Mosher, Lewis & Leahy, 1992) and non-parametric methods such as Low Resolution Electrical Tomography (LORETA; Pascual-Marqui, Michel & Lehmann, 1994), standardised Low Resolution Electromagnetic Tomography (sLORETA; Pascual-Marqui, 2002), Weighted Minimum Norms (WMN; Gorodnitsky & Roa, 1997) and Shrinking LORETA-FOCUSS (SLF; Lui et al., 2004). Parametric methods estimate dipole parameters based on *a priori* determined number of dipoles, which means that the results can be extremely dependent on the initial assumptions made (Grech et al., 2008). Non-parametric methods, on the other hand, make no *a priori* dipole location assumptions. Since there is limited research investigating gustatory EEG, we made no *a priori* assumptions and chose the non-parametric sLORETA technique. sLORETA is a tomographic method whereby localisation is inferred based on images of standardised current density and this is purported to have zero localization error (Pascual-Marqui, 2002). When compared with other non-parametric source localization techniques (e.g., WMN, SLF, LORETA) using a three-shell spherical head model registered to the Talairich human brain atlas (Talairach & Tournoux, 1988), sLORETA was shown to have the best performance in terms of localization error (see Grech et al., 2008, for a review).

2.5.5. Oscillatory activity: Event related de/synchronisations

When EPSPs summate into major depolarisations, a periodic sequence of afferent bursts showing sinusoidal potential fluctuations can sometimes result, which is known as an oscillation. The number of bursts per second is the frequency of an oscillation, for example a frequency of 50 Hz is 50 oscillations per second. Neural oscillations are generally considered within the rhythm, or frequency band in which they operate: delta-band rhythm ranges between 0 – 4 Hz; theta-band ranges from 4 – 7 Hz; alpha-band is 7 – 13 Hz; beta is 13 – 30 Hz and gamma is 30 – 80 Hz.

Neural synchrony refers to groups of neurons oscillating at the same frequency at the same time. When distinct populations of neurons oscillate at different frequencies they are desynchronised; when they oscillate at the same frequency they are synchronous. It is believed that the synchronisation of oscillatory neuronal firing represents a physiological coding mechanism that binds together spatially separated populations of neurons (Eckhorn et al. 1988; Gray et al. 1989; for a review, see Singer 1993). Changes in oscillatory synchronisation relative to the onset of a stimulus or event are known as event-related synchronisation (ERS) or event-related desynchronisation (ERD). ERS is generally associated with deactivations of cortical areas, except in the case of theta-band oscillations where ERS is associated with an increase in cortical activity. ERD, on the other hand, is interpreted as a correlate of an activated cortical area with increased excitability (Pfurtscheller, 2001). The strength of activity within these synchronised events is determined by the amplitude or power of the oscillation. This is dictated by the number of neurons in a population that fire during the burst. For example, if 40 out of a population of 200 neurons fire within the same burst, this will give a power of 20% and this value is what is calculated during ERD/S analysis.

Neuronal oscillations are considered part of a mechanism that modulates cognitive and sensory input and oscillations in different frequency bands are believed to be functionally distinct (Klimesch et al., 1996). For example, theta-band oscillations (4 – 7 Hz) are strongly linked with hippocampal activity and memory processes (e.g., Caplan et al., 2003; Doppelmayr et al., 2008; Klimesch et al., 1994; Klimesch et al., 1996, Klimesch et al., 1997; Klimesch et al., 1999; Klimesch et al., 2001a; Klimesch et al., 2006) and attention (Deiber et al., 2007; Gevins et al., 1997;

Gevins & Smith, 2000; Pennekamp et al., 1994; Sauseng et al., 2007), with increased theta-band activity associated with the encoding and retention of information. Alpha- (7 – 13 Hz) and beta-band oscillations (13 – 30 Hz), on the other hand, are commonly associated with attentional, motor and sensory networks; decreasing in amplitude (ERD) with cortical excitation, and increasing in amplitude (ERS) with cortical inhibition (e.g., Palva et al., 2005; Pfurtshceller & Lopes da Silva, 1999; Pfurtscheller, Neuper & Mohl, 1994; Sausing et al., 2005). Higher frequencies, such as gamma-band oscillations (30 – 80 Hz), originate from a number of cortical and subcortical regions and increases in gamma amplitude have been largely associated with higher order cognitive functions such as decision-making, motivation and short and long-term memory functions (see Bosman, Lansink & Pennartz, 2014, for a review). Figure 2.6 (b) illustrates a time-frequency representation (TFR) plot and the functionally distinct oscillatory frequency components.

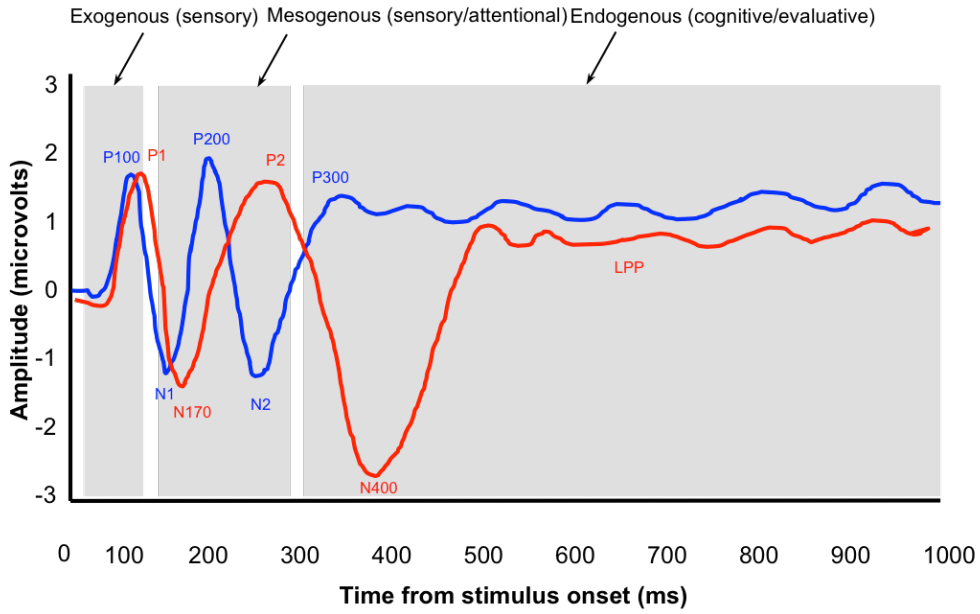

Fig. 2.6 (a). An illustrated example of a typical ERP plot (amplitude x time) and the ERP components described in Section 2.5.2. Grey boxes distinguish between exogenous, mesogenous and endogenous ERP signals.

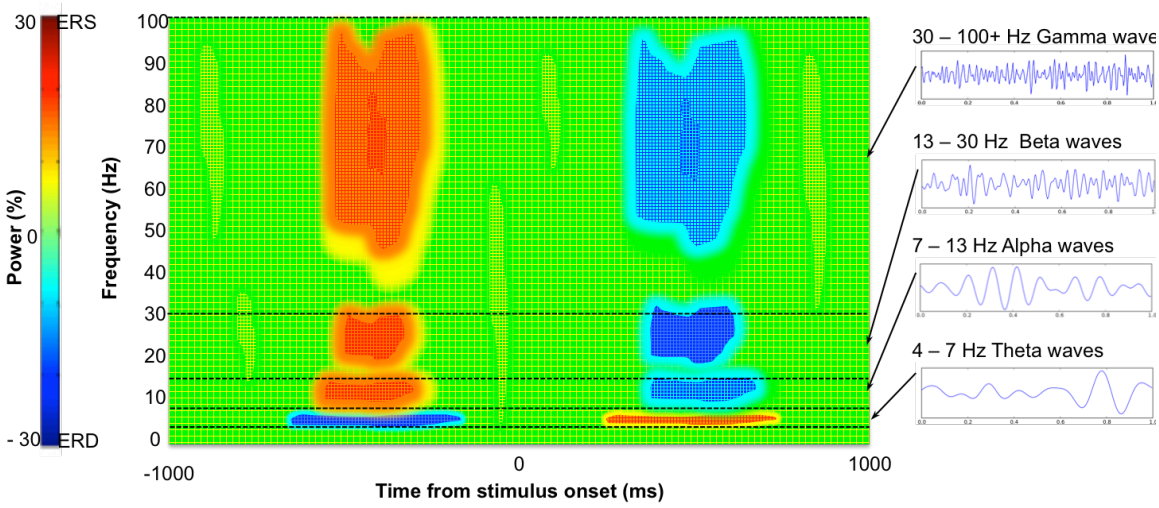

Fig. 2.6 (b). An illustrated example of a TFR plot (frequency x time) and the oscillatory components described in Section 2.5.5. The colour bar demonstrates the power of the frequency signal (%) with red associated with an increase in power (ERS) and blue with a decrease in power (ERD). The typical frequency waves are shown to the right of the TFR diagram**.**

Chapter Three: Dissociating the Gustatory Coding of Quality, Intensity and Hedonic Value

3.1. Abstract

The neural representation of the quality, intensity and hedonicity of gustatory stimuli remains largely uncharacterised. In this chapter, we investigated the differential electrophysiological processing of these separate characteristics of taste using a carefully designed stimulus set and a custom built gustometer. We examined ERPs, the source-localisation of ERPs and ERD/S in response to four taste qualities (salt, bitter, sweet and water), four levels of taste intensity (weak, medium, strong and neutral) and three levels of hedonicity (unpleasant, pleasant and neutral). Taste intensity was processed in early (60 – 140 ms) and late (1000 – 1500 ms) ERP activity generated from the PGC and inferior parietal lobe. Quality and hedonic attributes were coded in late (> 680 ms) ERP epochs in the same regions. However, the observed patterns of activity did not fit precisely with the psychophysical attributes of the tastes and indicated confounding influences of arousal as well as habituation. For ERD/S, taste intensity and hedonicity were represented by alpha- and beta-band ERD, and taste quality was distinguished by theta- and alpha-band ERD. The data revealed distinct differences in the coding of different taste intensities and were able to differentiate between each of the three levels of taste hedonicity. We conclude that ERD/S analysis may be the optimal method for reliably distinguishing between the processing of different taste characteristics. However, taste quality coding proved difficult to characterise.

3.2. Introduction

The neural representation of the quality, intensity and hedonicity of sensory stimuli has long been a central focus of sensory neuroscience. While much research has been devoted to visual and auditory modalities, less progress has been made in the chemosenses. In particular, the cortical processing of gustatory information in humans remains largely uncharacterised. Although it is understood that gustatory coding follows a path beginning with cell activity in peripheral taste fibres and extending across a network of primary and secondary gustatory processing structures (Frank et al., 1983; Hellekant et al.,1981; Katz et al., 2001; MacDonald et al., 2012; Ogawa et al., 1984; Scott and Plata-Salaman, 1999), the specific neural coding schemes of taste quality, intensity and hedonicity remain poorly understood.

One way to measure human sensory coding is the study of time-domain (ERP) and oscillatory (ERD/S) neural signals using EEG. A benefit of this analysis is that it can provide covert, online measurements of stimulus processing to determine which stage of processing is affected by a specific experimental manipulation (Luck, 2005). Using this method, previous studies have shown that human sensory information is generally parsed with analysis of simple, low-level physical attributes in the early stages of cortical processing (< 300 ms), with later stages (> 300 ms) being associated with the assembly of these features into a more complex percept (Goldstein, 2009), together with affective, reward or arousal characteristics (see Hajcak et al., 2010). Moreover, ERD/S analysis has determined that neural oscillations are associated with sensory processing in visual, auditory, somatosensory and olfactory domains (Basar et al., 2012; Eckhorn et al., 1988; Engel et al., 2001; Kayser et al., 2012; Laurent & Davidowitz, 1994; Nicolelis et al., 1995), with pleasant stimuli often associated with a left-lateralised alpha-band ERD responses (Balconi & Mazza, 2009; Davidson & Henriques, 2000; Waldstein et al. 2000).

Gustatory EEG studies, however, are rare. This is due to the fact that they are notoriously difficult to conduct (see Chapter Two, section 2.4, for a full review of this issue). Despite the challenges in obtaining gustatory EEG data, however, some studies examining gustatory intensity, hedonic and quality coding have yielded highly relevant outcomes. For example, intensity dependent amplitude shifts have been observed in both early (P1: e.g., Hummel et al., 2010; Mizoguchi et al., 2002;

Ohla et al., 2010; N1: e.g., Ianilli et al., 2014; Mizoguchi et al., 2002) and late (P2: e.g., Hummel et al., 2010; LPP: e.g., Ianilli et al., 2014; Mizoguchi et al., 2002) ERP components, and localised to the insula and opercula regions in the PGC (Ianilli et al., 2014; Mizoguchi et al., 2002; Ohla et al., 2010) as well as parietal somatosensory cortices (e.g., Ohla et al., 2010). In addition, pleasant tastes have been associated with increases in P1 and P3 amplitudes compared with water (e.g., Franken et al., 2011) and a left-hemispheric alpha-ERD (Fox & Davidson, 1986; Morinushi et al., 2000); with unpleasant tastes evoking a right-lateralised theta-ERD in one study (e.g., Toth et al., 2004). Most recently, Crouzet et al. (2015) reported that taste quality coding began as early as 150 ms after stimulus onset, with signal increases for different tastants distinguished by their latency.

Taken together, current gustatory EEG investigations go some way to support a model of sensory processing similar to those in other modalities, in that low-level stimulus features (i.e., taste intensity and quality) appear to be processed earlier and hedonic components later. In addition, the research suggests that pleasant gustatory stimulation results in alpha-band ERD. However, with the very limited research in this area, findings are often inconsistent or inconclusive. In particular, the coding of taste quality is considerably disputed (see Spector and Travis, 2005 for a full review of this issue). Furthermore, imaging studies have found that neural areas implicated in the processing of taste quality (e.g., the insula; Bender et al., 2009; Pritchard et al., 2005), have also been implicated in taste intensity (Grabenhorst et al., 2008) and hedonicity processing (Small et al., 2003).

In fact, a general caveat in taste processing studies is that these investigations have examined quality, intensity and hedonic characteristics either as a secondary measure in ERD/S studies (e.g., Fox et al., 1986; Morinushi et al., 2000; Toth et al., 2004) or entirely separately in the ERP studies (e.g., Franken et al., 2011; Hummel et al., 2010; Grabenhorst et al., 2008), and we must be mindful that there exists interactions between them; to the extent that participants are often unable to rate the separate characteristics independently of each other (Pfaffmann, 1980). With these characteristics being so intertwined, it is important to attempt to differentiate their processing through careful consideration of the stimuli set (e.g., Sadacca et al., 2012).

The current study examined the temporal coding of taste quality, intensity and hedonicity. Using a carefully devised stimulus set, we compared ERP, source-localised ERP latencies and ERD/S responses to taste stimuli comprising four taste qualities (salt, bitter, sweet and water), four levels of taste intensity (weak, medium, strong and neutral) and three levels of hedonicity (unpleasant, pleasant and neutral). The study is the first to employ an extensive range of taste stimuli by which to compare human EEG responses evoked by these separate taste characteristics. Nevertheless, the total number of repetitions of each stimulus was confined by practical limitations on the overall duration of testing, with a consequent restriction on our ability to discern fully articulated ERP components. However, we refer to the temporal effects as 'ERPs' despite the absence of typical peaks.

3.3. Materials and Methods

3.2.1. Participants

Twenty-nine participants (6 male) aged 19–33 years (M = 24.18, SD = 3.91) took part in the EEG study. Data from seven participants were removed from analyses due to substantial noise in their EEG recordings. All participants were pre-screened and were non-smokers, had no taste disorders and were not taking medications or suffering illnesses that interfered with their gustatory or olfactory perception. All participants gave informed consent and all work was carried out in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki), and was approved by the University of Liverpool Ethics Committee.

3.2.2. Design

A 4 × 4 × 3 within-subjects design was employed. The independent variables were taste quality (salt, bitter, sweet, water), taste intensity (weak, medium, strong, neutral) and taste hedonicity (unpleasant, pleasant, neutral). The dependent variables were ERP amplitude, current densities at ERP latencies and ERD/S power.

3.2.3. Taste stimuli

The selection of the taste stimuli for each condition is described in Chapter Two (section 2.3). The tastants for this study included NaCl 0.05 M (salt 1), 0.1 M (salt 2), 0.3 M (salt 3); 0.0003 M QHCl (bitter), 0.3 M sucrose (sweet), with distilled

water (water) added as a neutral control. These were grouped into conditions of quality (water, bitter, sweet, salt), intensity (neutral, weak, medium, strong), and hedonicity (pleasant, unpleasant). The conditions individual tastant formed are presented in Table 3.1, along with mean (±SD) ratings of intensity (gLMS; Bartoshuk et al., 2004) and pleasantness (LAM; Schutz & Cardello, 2001) taken from participants in the preliminary study (1) and from different participants prior to the EEG investigation (2). ANOVA revealed no significant differences between results from the preliminary study (1) and the current data (2) for intensity (p = .942) and pleasantness (p = .881) for all tastes (Table 3.1). In addition, water (neutral) was rated as significantly less intense than the weak, medium and strong conditions (*MD*s > 11.72, *SE*s < 4.87, *p*s < .019), and differed in pleasantness from both positive and negative conditions (*MD*s > 9.18, *SE*s < 4.61, *p*s < .049). All participants were able to correctly identify all taste qualities.

Table 1

Taste	Grouping			Mean Rating (±SD)			
	Quality	**Intensity**	**Hedonicity**	**Intensity (1)**	**Intensity (2)**	**Pleasantness (1)**	**Pleasantness (2)**
Salt 1 (NaCl 0.05 M)	Salt	Weak	Unpleasant	16.41 (26.3)	11.68 (10.34)	-4.43 (10.25)	-5.23 (10.64)
Salt 2 (NaCl 0.1 M)	Salt	Medium	Unpleasant	21.22 (11.47)	25.40 (15.23)	-8.18 (12.20)	-9.45 (13.45)
Salt 3 (NaCl 0.3 M)	Salt	Strong	Unpleasant	33.08 (16.10)	35.42 (18.44)	-10.41 (13.50)	9.60 (16.52)
Bitter (QHCl 0.0003 M)	Bitter	Strong	Unpleasant	27.24 (13.60)	30.31 (21.55)	-19.50 (11.56)	-19.06 (12.81)
Sweet (Sucrose 0.3 M)	Sweet	Strong	Pleasant	27.94 (14.75)	25.35 (11.50)	19.41 (26.36)	18.70 (31.56)
Water	Water	Neutral	Neutral		0.052 (15.87)		-1.65 (21.30)

Table 3.1. The designation of each taste solution into each taste quality, intensity and hedonicity condition. Mean intensity and pleasantness ratings (± SD) for both the preliminary (1) and current (2) study are also shown.

3.2.4. Stimuli presentation

The stimuli were presented using a computer-controlled gustometer (described in full in Chapter Two, section 2.4.2). This comprised eight diaphragmatic electronically-controlled pumps (KNF STEPDOS FEM03.18RC, Verder, Vleuten, The Netherlands) which were operated using Psychopy open-source software (Pierce, 2007). The pumps delivered solutions via separate tubing to a common, 8-component manifold with an inline check valve to prevent cross-contamination. The software was also used to interface with a monitor to provide instructions to the participants.

The taste solutions were administered to the centre of the tongue via 1.6 mm internal diameter Teflon tubing clamped to a head-rest. Each 1 ml taste sample was administered over 2 seconds at a flow rate of 30 ml min. Technical measurements prior to the study established the rise time to be less than 0.02 seconds from the serial port signal being returned to the software. Each participant was required to hold the solution in his or her mouth for 3 seconds whilst remaining still, before swallowing. Each tasting was followed by a 4-second (2 ml) distilled water rinse and swallow. At the end of each trial, there was a rest period which allowed for an ISI ranging between 20 – 30 seconds, so controlling for habituation and adaptation (Evans et al., 1993). A new trial was only initiated when EMG data, displayed on a computer monitor, showed no signs of swallowing motions. Given this method of delivery, concomitant somatosensory influences (see Ohla et al., 2011) were unavoidable, however, all taste stimuli were presented in an identical matter therefore any differences in activations between the tastes themselves may be attributed to a gustatory effect.

The order of the taste samples was randomised, with steps taken to ensure that the same taste sample was never presented on two consecutive trials. Overall, each of the six stimuli was repeated 30 times (180 trials, separated into 6 blocks of 30 trials). Breaks were taken between blocks. Thirty stimulus repetitions were selected based on a recommendation of 30 – 40 trial repeats for gustatory EEG (Mizoguchi et al., 2002). We chose the lower end of this suggestion to reduce testing durations and offset this with an increased population of participants (based on n = 16 recommended for EEG analysis). Figure 3.1 provides a schematic representation of the experimental procedure and presentation.

3.2.5. Additional measures

Participant BMI, taster status (Tepper, 2001) and appetite (Flint et al., 2000; Rolls et al., 1999) measures were taken and participants were formed into groups of normal, over-weight and obese; non-taster, medium-taster and super-taster; and low-and high hunger (median split). BMI (*p*s > .44) and taster status (*p*s > .063) were found to have no impact on EEG data so are not discussed further.

3.2.6. Procedure

Participants arrived at the laboratory having been requested to consume no food or drink (other than water) for 2 hours before the session. Participants tasted and rated each of the tastes three times in a randomised order for quality, intensity and pleasantness to enable comparisons with the results of the preliminary study. The EEG equipment was fitted to the participant who was then seated in the experimental chamber. After completing a practice trial, the main experiment began, with stimuli delivered as described above. Overall, the experiment took 2 - 2.5 hours to complete, depending on the length of the breaks taken between blocks of trials.

3.2.7. Electrophysiological measures: ERP, sLORETA and ERD/S

The data were recorded using a BioSemi Active-Two amplifier system (BioSemi BV, Amsterdam, Netherlands), with 64 scalp electrodes arranged according to the International 10–20 System (Oostenveld & Praamstra, 2001) and placed in an elastic cap. Common Mode Sense (CMS) and Driven Right Leg (DRL) electrodes were used as a reference and ground, respectively. The EEG was continuously recorded at 512 Hz with a band pass filter of 0.001 - 100 Hz. Two external EMG electrodes were placed over the massiter muscles to detect swallowing movements, and were sampled at 512 Hz. The EMG data were used to initiate trials based on visual inspection (see above) and were not analysed further.

The EEG data were analysed offline using EEGLAB (Delorme & Makeig, 2009), sLORETA (Pascual-Marqui, 2002) and FIELDTRIP (Oostenveld et al., 2011) toolboxes in combination with custom Matlab scripts. Trials in which the gustometer failed to operate correctly were excluded from analysis. Each participant's recording was average-referenced to all electrodes, low pass filtered at 30 Hz and then down-sampled to 128 Hz to reduce file size. The continuous data were then segmented into -1000 ms to 3000 ms epochs. Bad channels identified through visual inspection and

kurtosis (threshold = 5) were removed and interpolated (Delorme & Makeig, 2009). An independent components analysis (ICA) was used to identify and extract ocular and other muscular artefacts (Jung et al., 2000). Data were divided into 64 independent components and an average of 14.5 (*SD* = 10.66) noise components were removed for each participant. Following ICA, trials that exceeded ±100 μV at any electrode were removed. The number of trials removed for each condition was determined and any participant with more than 50% of trials removed for one or more conditions was excluded from the final analysis (n = 7). The final mean number of trials remaining (± *SD*) included 21.0 (± 4.22) for salt 1, 21.11 (± 3.75) for salt 2, 20.77 (± 3.43) for salt 3, 19.77 (± 3.65) for bitter, 20.81 (± 4.11) for sweet and 26.77 (± 2.16) for water. There were no significant differences in the number of trials remaining for each taste (*p*s >. 07).

For sLORETA, the electrode coordinates were created from the 64 electrode locations using the original recording montage. A transformation matrix was created using the electrode coordinates. The averaged waveforms were converted and saved into sLOR values for each condition and subject. Computations were made in a realistic head model (Fuchs et al., 2002), using the MNI152 template (Mazziotta et al., 2001), with the three-dimensional solution space restricted to cortical gray matter. The intracerebral volume is partitioned in 6239 voxels at 5 mm spatial resolution. Anatomical labels as Brodmann areas are reported using an appropriate correction from MNI to Talairach space (Brett et al., 2002). Thus, sLORETA images represent the electric activity at each voxel in neuroanatomic Talairach space (Talairach & Tournoux, 1988) as the squared standardised magnitude of the estimated current density.

As oscillations are not phase-locked, but time-locked to a stimulus, they may be removed by averaging across trials. It was therefore necessary to compute ERD/S power on a trial-by-trial basis before averaging. On each trial, the data were filtered to a 2 – 30 Hz bandwidth with a -500 – 0 ms baseline. Raw data were then convoluted with a Hanning-tapered wavelet comprising four cycles at each frequency. Relative power was computed as a proportion change from the baseline and wavelets were positioned at increments separated by 50 ms through the raw data.

3.2.8. Statistical analysis

We performed three types of analysis: the standard time-domain averaging technique to measure ERPs, sLORETA to examine the origins of the ERP effects and a wavelet-based time-frequency representation (TFR) to analyse underlying neural oscillations in the form of ERD/S.

ERP analysis

To evaluate mean ERP differences between the different levels of intensity, quality and hedonicity, the EEG data were analyzed using one-way repeated measures ANOVA for each condition at each electrode and each time point in the range from -200 to 1500 ms. The statistical significance for the ERP analysis was evaluated using the permutation method (Maris & Oostenveld, 2007) in the EEGLAB v. 9 program package (http://sccn.ucsd.edu/eeglab/) involving 500 permutations. This analysis controls for the Type I error associated with the large number of components and time points. A 95% confidence level was always employed. This data driven method allows for complete objectivity in the selection of components that contribute to EEG data.

Electrodes showing statistically significant differences in each condition were combined into clusters and averaged. Individual ERPs for each cluster were evaluated for each participant, and subjected to a series of within-subjects ANOVAs with the factors: intensity (neutral, weak, medium, strong), quality (water, salt, bitter, sweet) and hedonicity (neutral, pleasant, unpleasant).

sLORETA analysis

When significant ERP components were identified, sLORETA was used to compute the cortical three-dimensional distribution of the current density at each significant latency identified for intensity, quality and hedonicity. By this method, the maximum of the current density obtained at each significant latency was taken as the source of the particular component. We calculated sLORETA images for each ERP in the time frame -200 – 1500 ms post-stimulus. sLORETAs for each source were obtained for each participant and subjected to a series of within-subjects ANOVAs with the factors: intensity (neutral, weak, medium, strong); quality (water, salt, bitter, sweet) and hedonicity (neutral, pleasant, unpleasant).

ERD/S analysis

For the ERD/S analysis, the percentage of power decrease or increase was calculated using the ERD%=(A−R)/R∗100 expression (Pfurtscheller & Aranibar, 1979), where A (Absolute) is the power within the frequency band of interest in the activity period and R (Reference) is the preceding baseline or reference period. Using one-way repeated measures ANOVA for each condition at each electrode and frequency band (theta, 4 – 7 Hz; alpha, 7 – 13 Hz; beta, 13 – 30 Hz) between -1000 and 3000 ms. The statistical significance was evaluated using the permutation method (Maris & Oostenveld, 2007) involving 500 permutations. Electrodes showing statistically significant differences in each condition were combined into clusters and averaged. Individual ERD/S at each cluster were evaluated for each participant, and subjected to a series of within-subjects ANOVAs with the factors: intensity (neutral, weak, medium, strong); quality (water, salt, bitter, sweet) and hedonicity (neutral, pleasant, unpleasant).

Post hoc analyses using pairwise comparisons and Bonferroni corrections were conducted for each EEG analysis when significant effects occurred. Greenhouse-Geisser corrections were applied when statistical assumptions were not met. Where multiple significant effects occurred, results were collated to show the smallest mean difference, greatest standard error and greatest *p* values respectively (*MD*s >, *SE*s <, *p*s <). Effect sizes (ES) represent the partial η^2 value.

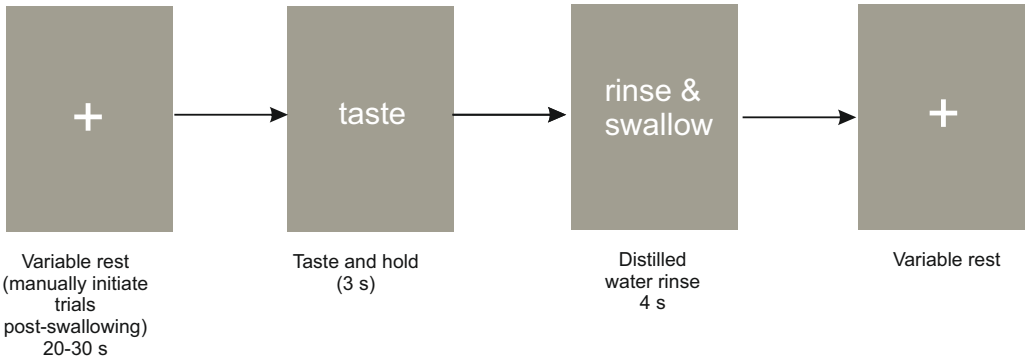

Fig 3.1. A schematic diagram of the experimental procedure showing the timeline of events within a trial and the cue the participant observed on the monitor at each event. Each trial began with a 20 – 30 s rest period after which the taste was administered and held orally for 3 s, followed by a 4 s distilled water rinse**.**

3.3. Results

3.3.1. ERP Analysis

As Figure 3.2 illustrates, no fully articulated ERP waveform could be observed. However, distinct temporal and regional differences in ERP magnitude were apparent for taste intensity, quality and hedonicity (Figure 3.3). The principal effects are summarised below for each factor.

Taste intensity

Our analysis revealed significant effects of taste intensity on ERP amplitude in the right parietal-occipital region (P2, P4, P6, P10, Po8, O2) between 60 – 140 ms, F (2.05, 43.14) = 4.50, p < .016, ES = .18, and from 1000 – 1500 ms, F (3, 63) = 7.03, p <. 001, ES = .25.

As revealed by the ERP plot and topographic maps [Figure 3.3 (a, b)] between 60 – 140 ms, both weak and strong tastes evoked greater positive amplitudes than either medium or neutral tastes, which themselves produced little change. Statistically, when corrections were applied (p < .008), only responses to weak and medium tastes differed (MD = 0.86, SE = 0.22, p = .005).

Interestingly, taste interacted with ratings of hunger here, F (3, 60) = 7.06, p < .001, ES = .26, where weak taste intensities evoked greater amplitudes than neutral tastes, but only in those who reported lower hunger (MD = 1.29, SE = 0.24, p = .002), and not in those who reported greater hunger (p > .99).

The influence of intensity continued in the later epoch (1000 – 1500 ms), F (3, 63) = 6.71, p < .001, ES = .24, where we can again see from the ERP plots and topographic maps [Figure 3.3 (a, c)] a clear separation of the positive amplitudes evoked by weak and strong tastes, relative to the negligible amplitudes evoked by medium and neutral stimuli. With corrections applied, only neutral and strong tastes show significant differences from each other (MD = 1.92, SE = 0.48, p = .004).

An effect of taste intensity on ERP amplitude also emerged in the right fronto-central region (Af4, Fz, F2, F4, Fc4, Fcz, Cz) between 1000 – 1500 ms, F (2.04, 42.80) = 7.06, ps = .002, ES = .25. As can be seen from Figure 3.3 (d, e), both medium and neutral tastes evoked negative amplitudes of a similar magnitude that were greater than those following either weak or strong stimuli. With corrections

applied, only neutral and strong tastes significantly differed from each other (*MD* = 1.72, *SE* = 0.33, *p* = .002).

Taste quality

As can be seen in Figure 3.3 (f, g), significant effects of taste quality on ERP amplitude were apparent in the right parietal-occipital region (P5, P7, Po3, O1, Iz, Oz) between 680 – 780 ms, *F* (3, 63) = 8.82, *p*s <. 001, ES = .30, and from 1200 – 1500 ms, *F* (2.03, 42.62) = 6.45, *p* = .003, ES = .24.

From 680 – 780 ms, all tastes evoked positive amplitudes, with each inducing greater amplitudes than responses from water [Figure 3.3 (f, g)]. Bitter evoked the greatest positive amplitude, followed by sweet, salt and finally water. Statistically, with corrections applied (*p* < .008), bitter and water significantly differ from each other (*MD* = 2.04, *SE* = 0.48, *p* = .002).

In the later epoch [1200 – 1500 ms; Figure 3.3 (f, h)], all taste qualities evoked similarly greater positive amplitudes compared with water, which showed little change. Statistically, water differed from both bitter and sweet tastes (*MD*s > 2.06, *SE*s < .57, *p*s < .007), but not salt (*p* = .11)

Taste hedonicity

As summarised in Figure 3.3 (i), significant effects of taste hedonicity were apparent in the right fronto-central region (Af4, Fz, F2, F4, Fc4, Fcz, Cz) from 600 – 1500 ms, *F* (2, 42) = 16.84, *p* < .001, ES = .45. Compared to neutral taste, which evoked little change, both pleasant and unpleasant tastes evoked greater negative amplitudes (*MD*s > 1.20, *SE*s < 0.24, *p*s < .001), which did not differ from each other (*p* = .70).

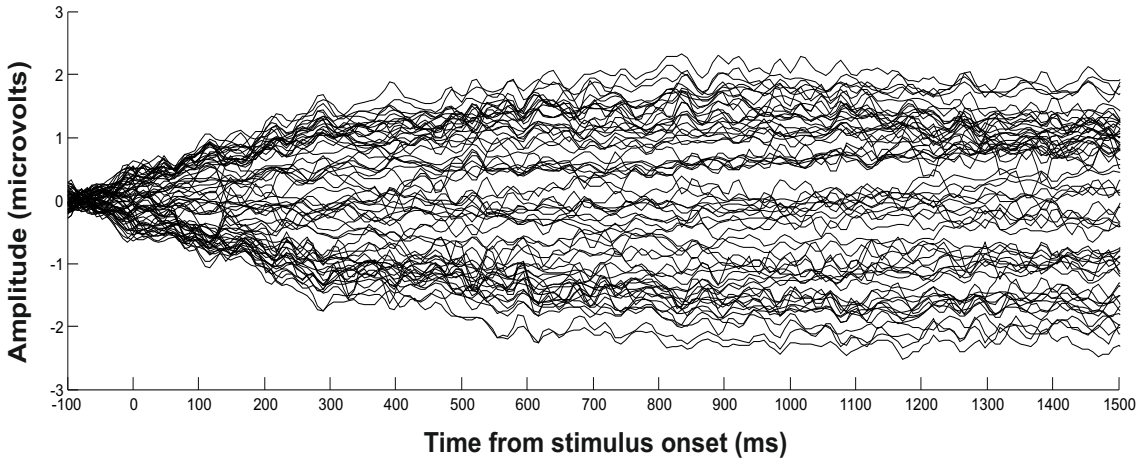

Fig. 3.2. Butterfly plot demonstrating the average ERP waveform across conditions for each electrode (separate lines) from the onset of the tastant**.**

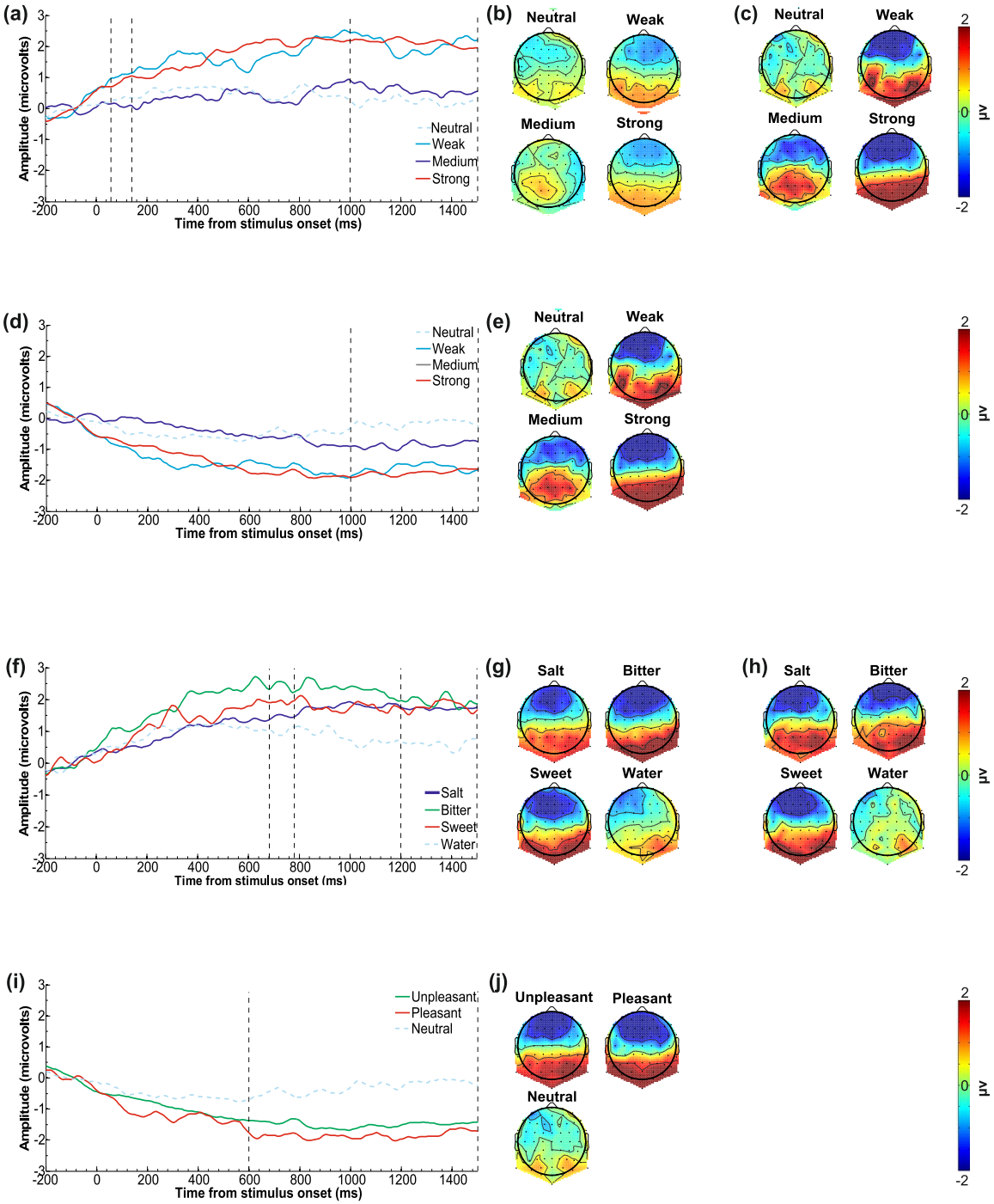
(a)
Amplitude (microvolts)
Time from stimulus onset (ms)
Neutral
Weak
Medium
Strong
(b)
Neutral
Weak
Medium
Strong
(c)
Neutral
Weak
Medium
Strong
μV
(d)
Amplitude (microvolts)
Time from stimulus onset (ms)
Neutral
Weak
Medium
Strong
(e)
Neutral
Weak
Medium
Strong
μV
(f)
Amplitude (microvolts)
Time from stimulus onset (ms)
Salt
Bitter
Sweet
Water
(g)
Salt
Bitter
Sweet
Water
(h)
Salt
Bitter
Sweet
Water
μV
(i)
Amplitude (microvolts)
Time from stimulus onset (ms)
Unpleasant
Pleasant
Neutral
(j)
Unpleasant
Pleasant
Neutral
μV

Fig. 3.3. Temporal ERP amplitude plots with vertical dashed lines indicating intervals with significant main effects of condition, and topographic head plots with dashed circles indicating the regions of interest (ROI), with colour bars representing amplitude (μV). (a) Significant ERP effects of taste intensity in the left parietal-occipital region (P2, P4, P6, P10, Po8, O2) between 60 – 140 ms (b) and 1000 – 1500 ms (c). (d) Significant ERP amplitude effects of taste intensity in the right fronto-central region (Af4, Fz, F2, F4, Fc4, Fcz, Cz) between 1000 – 1500 ms (e). (f) Significant ERP effects of taste quality in the right parietal-occipital region (P5, P7, Po3, O1, Iz, Oz) between 680 – 780 ms (g) and 1200 – 1500 ms (h). (i) Significant ERP effects of taste hedonicity in the right fronto-central region (Af4, Fz, F2, F4, Fc4, Fcz, Cz) from 600 – 1500 ms (j).

3.3.2. sLORETA Analysis

Figure 3.4 illustrates the sLORETA source estimates of each of the ERP latencies and the current densities for each condition. The principal sources are summarised below for each factor.

Taste Intensity

During the early (60 – 140 ms) taste intensity latency, a cluster of activation was observed in right parietal operculum [TAL, $x = 64$, $y = -23$, $z = 38$; Figure 3.4 (a)]. There were no significant differences in the current densities from this region for the taste intensity conditions [$p = .079$; Figure 3.4 (b)]. During the later latency (1000 – 1500 ms) activations were observed in right inferior parietal lobule [TAL, $x = 65$, $y = -31$, $z = 47$; Figure 3.4 (c)], but there were no differences observed in current densities for the taste intensity conditions [$p = .079$; Figure 3.4 (d)].

Taste quality

During the 680 – 780 ms latency for taste quality, a cluster of activation was observed in the right parietal operculum [TAL, $x = 64$, $y = -23$, $z = 38$; Figure 3.4 (e)], with densities most enhanced for sweet tastes. However, differences in the current densities at this location for the taste quality groups failed to reach significance [$p = .071$; Figure 3.4 (f)]. In the later latency (1200 – 1500 ms), activations were found in the right inferior parietal lobule [TAL, $x = 65$, $y = -31$, $z = 47$; Figure 3.4 (g)], but no differences in taste responses to the tastant qualities emerged [$p = .341$; Figure 3.4 (h)].

Taste hedonicity

During the 600 – 1000 ms latency for taste hedonicity, activations were found in the right inferior parietal lobule [TAL, $x = 65$, $y = -31$, $z = 47$; Figure 3.4 (i)]. Although slightly posterior to the observed ERP effects, there was a significant effect of hedonicity here, F (11.54, 32.41) = 13.19, $p < .001$, ES = .22 [Figure 3.4 (i)]. Sweet tastes evoked the greatest current density, followed by neutral and unpleasant taste. Statistically, the current density evoked by unpleasant tastes significantly differed from both pleasant and neutral tastes ($MDs > 0.12$, $SEs < 0.39$, $ps < .013$). Responses

to sweet also differed from responses to neutral (*MDs* > 0.16, *SEs* < 0.06, *ps* < .042), until Bonferroni corrections were applied ($p < .017$).

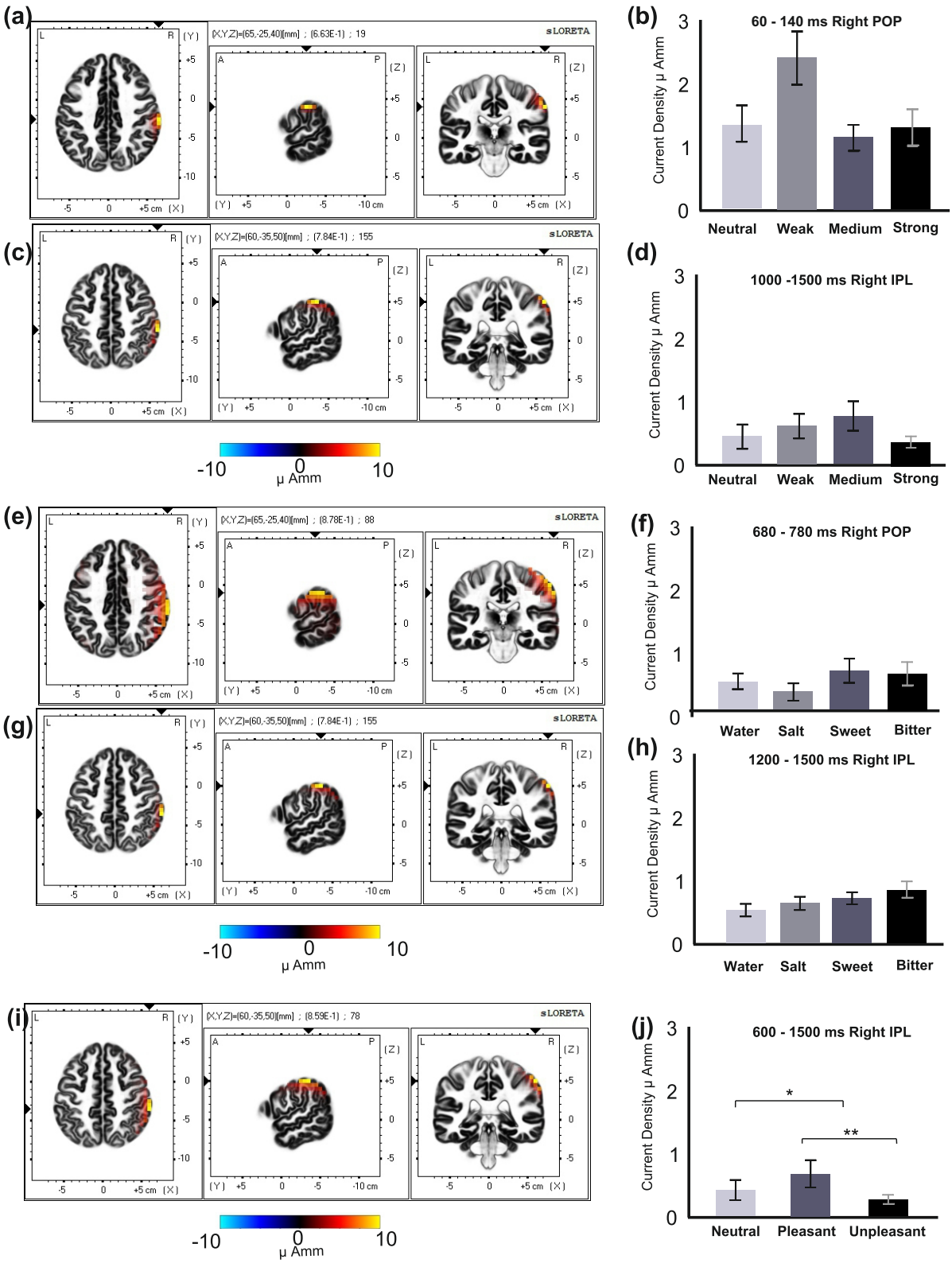
(a)
(b)
60 - 140 ms Right POP
Current Density µ Amm
Neutral
Weak
Medium
Strong
(c)
(d)
1000 -1500 ms Right IPL
-10
0
10
µ Amm
(e)
(f)
680 - 780 ms Right POP
Water
Salt
Sweet
Bitter
(g)
(h)
1200 - 1500 ms Right IPL
(i)
(j)
600 - 1500 ms Right IPL
*
**
Pleasant
Unpleasant
sLORETA

Fig. 3.4. sLORETA imaging results displaying maximum current density at each ERP latency for the grand mean results, with colour bars representing current density (μ Amm) and bar charts showing the current density for each condition, with error bars indicating standard error. (a) sLORETA image showing the maximum current density at 60 – 140ms located in the right parietal operculum. (b) Bar chart showing the mean current density at this location and latency for each taste intensity condition. (c) sLORETA image showing the maximum current density between 1000 – 1500 ms located in the right inferior parietal lobe. (d) Bar chart showing the mean current density for each taste intensity condition at this location and latency. (e) sLORETA image showing the maximum current density between 680 – 780 ms located in the right parietal operculum. (f) Bar chart showing the mean current density at this location and latency for each taste quality condition. (g) sLORETA image showing the maximum current density between 1200 – 1500 ms located in the right inferior parietal lobe. (h) Bar chart showing the mean current density at this location and latency for each taste quality condition. (i) sLORETA image showing the maximum current density between 600 – 1500 ms located in the right inferior parietal lobe. (j) Bar chart showing the mean current density at this location and latency for each taste hedonicity condition. Asterisks indicate significant differences: * $p < .05$; ** $p < .01$.

Note – the sLORETA images show the MNI coordinates for each effect, these have been converted to Taliarach coordinates in the results section (Brett et al., 2002)

3.3.3. ERD/S analysis

As summarised in the time-frequency spectrographs and topographic maps in Figure 3.5, distinct differences in ERD/S were apparent for taste intensity, quality and hedonicity. The principal effects are summarised below for each factor in relation to alpha-, beta- and theta-band oscillations.

Taste intensity

From 1.5 – 2.5 s after stimulus onset, significant effects of taste intensity on alpha-band oscillations (7 – 13 Hz) were evident in the central-parietal region (C1, Cp5, Cp3, Cp1, P1, P3, Cpz), F (3, 63) = 11.0, p < .001, ES = .34. From Figure 3.5 (a, b) it can be seen that medium and strong tastes evoked alpha-band ERD, whereas weak and neutral tastes showed alpha-band ERS or only minimal activity. Statistically, responses to medium and strong tastes show no differences (p = .465) and neither did responses to weak and neutral (p = .452). However, responses to weak tastes differed from responses to both medium and strong ones (*MD*s > 0.18, *SE*s < 0.46, *p*s < .003).

The analysis also revealed significant effects of taste intensity on beta-band activity (13 – 30 Hz) between 1.8 – 2.2 s in a smaller cluster of electrodes in the same central-parietal region (C1, C3, Cp3, Tp7), F (3, 63) = 7.57, p < .001, ES = .27. As evident in Figure 3.5 (a, c), beta-band ERD was greatest for medium and strong stimuli compared with weak and neutral tastes (*MD*s > 0.08, *SE*s < 0.03, *p*s < .017), however, this effect disappears when applying Bonferroni corrections (p < .008).

Taste quality

Between 1.8 – 2.2 s, taste quality exerted significant effects on theta-band oscillations (4 – 7 Hz) in the right fronto-central region (F5, Fc3, Fc1, C1, C5, Fcz, Cz) between 1.8 – 2.2 s, F (3, 63) = 4.07, p = .01, ES = .16 [Figure 3.5 (d, e)]. All tastes evoked centralised theta-band desynchronization, which was greatest for bitter tastes (*MD*s > 0.07, *SE*s < 0.04, *p*s < .04). However, this effect did not reach significance when applying Bonferroni corrections (p < .008).

Over the same interval, there was also a significant effect of taste quality observed on alpha-band activity (7– 13 Hz) in the left central-parietal region (C3, C5, Cp1, Cp3, Cp5, Tp7, P1, P3, P5), F (3, 63) = 5.72, p = .002, ES = .21. In contrast to the effects on theta-band oscillations, sweet tastes evoked the greatest alpha-band ERD [Figure 3.5 (d, f)], differing from salt and water (*MD*s > 0.11, *SE*s <

0.06, *p*s < .014), but not bitter (*p* = .071). However, the effects did not retain significance when applying Bonferroni corrections.

Taste hedonicity

Significant effects of taste hedonicity on alpha-band oscillations were observed in the left central-parietal region (C3, C5, T7, Cp1, Cp3, Cp5, Tp7, P1, P3, P5, P7) between 1.8 – 2.2 s, F (3, 63) = 7.58, p = .002, ES = .27 [Figure 3.5 (g, h)]. Pleasant tastes evoked the greatest alpha-band ERD here; this effect differed significantly from responses to unpleasant and neutral tastes, (*MD*s > 0.08, *SE*s < 0.05, *p*s < .012).

A different effect of taste hedonicity on beta-band oscillations (13 – 30 Hz) emerged in the left parietal-occipital region (P1, P5, P7, Po3, Oz, Poz) between 1.9 – 2.1 s, F (3, 63) = 4.02, p = .025, ES = .16. While pleasant tastes appeared to evoke the greatest desynchronisation here [Figure 3.6 (f, i)], this effect did not differ significantly from responses to unpleasant tastes (p = .597). However, responses to both pleasant and unpleasant stimuli differed from those after neutral stimuli, which evoked minimal change (*MD*s = 0.10, *SE*s = 0.49, *p*s = .05), although when corrections are applied this effect fails to maintain significance (p < .0125).

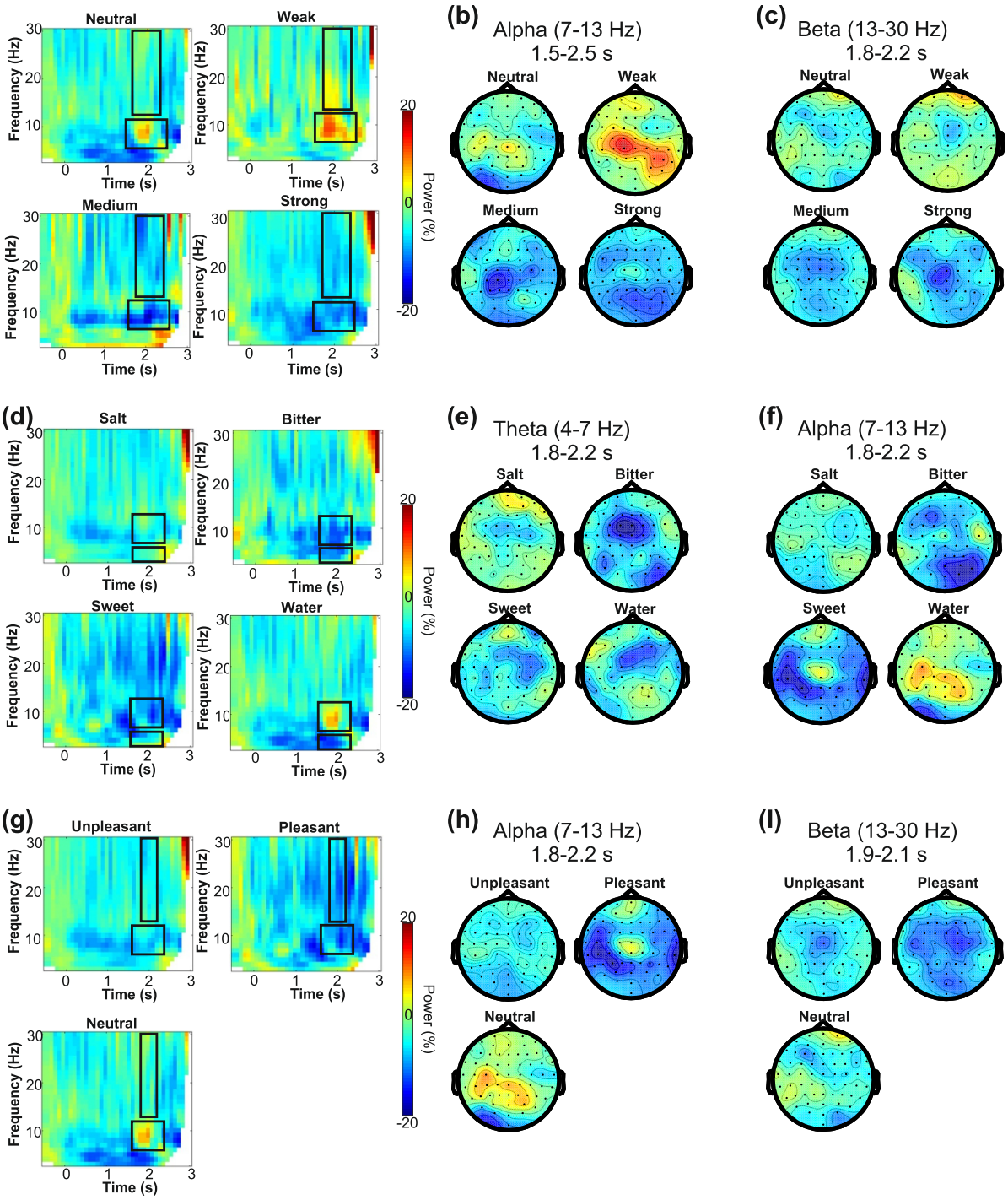
Neutral
Weak
Medium
Strong
Salt
Bitter
Sweet
Water
Unpleasant
Pleasant
Frequency (Hz)
Time (s)
Power (%)
(b) Alpha (7-13 Hz) 1.5-2.5 s
(c) Beta (13-30 Hz) 1.8-2.2 s
(d)
(e) Theta (4-7 Hz) 1.8-2.2 s
(f) Alpha (7-13 Hz) 1.8-2.2 s
(g)
(h) Alpha (7-13 Hz) 1.8-2.2 s
(I) Beta (13-30 Hz) 1.9-2.1 s

Fig. 3.6. Time-frequency spectrographs (TFR plots) across all electrodes depicting the power (%) and frequency of oscillations with significant effects indicated by boxes, and topographic head plots for each effect observed in the ERD/S analysis with dashed circles indicating the ROI. (a) Effects of taste intensity on (b) alpha-band oscillations between 1.5 – 2.5 s and (c) beta-band oscillations between 1.8 – 2.2 s. (d) Effects of taste quality on theta-band oscillations between (e) 1.8 – 2.2 s and (f) alpha-band oscillations between 1.8 – 2.2 s. (g) Effects of taste hedonicity condition on alpha-band oscillations between (h) 1.8 – 2.2 s and (i) beta-band oscillations between 1.9 – 2.1 s.

3.4. Discussion

The present chapter examined concurrent ERP, ERP source-localisation and ERD/S responses to the quality, intensity and hedonicity of tastants using a novel and diverse stimulus set. The EEG data were analysed across all electrodes, time points and various frequency bands relative to the onset of tastants, in order to begin to provide an understanding of the temporal and oscillatory processing of these gustatory characteristics. The ERP and source-localisation data revealed that taste intensity is distinguished at early (60 – 140 ms) and late (1000 – 1500 ms) stages of gustatory processing and were localised to primary gustatory cortices and inferior parietal regions respectively. Hedonicity was represented in late (> 600 ms) epochs and was localised to the inferior parietal lobule. The oscillatory data showed that taste intensity and hedonicity are represented with alpha (7 – 13 Hz) and beta-band (13 – 30 Hz) ERD. Effects of taste quality were obtained but specific differentiation of different tastes was not observed. These data go some way to support current models of sensory processing and extend those models to the gustatory domain.

Taste intensity

The ERP findings indicate that intensity was processed early (60 – 140 ms) in left parietal regions and later (1000 – 1500 ms) in left parietal and right frontal regions. Although the amplitude of the ERP responses to the different taste intensities did not always fit with the psychophysical responses to the tastants (discussed later), strong tastes evoked the greatest amplitude changes and neutral tastes evoked the smallest. The early effect fits well with the notion that early perceptual processing is dedicated to the analysis of simple stimulus attributes (Goldstein, 2009). Moreover, this effect supports the early intensity-dependent ERP differences previously reported (e.g., Hummel et al., 2010; Mizoguchi et al., 2002; Ohla et al., 2010) and suggests that the differences found by Franken et al. (2011) for pleasant and water tastes may, in fact, represent intensity rather than hedonicity. Interestingly, the early intensity effects interacted with hunger ratings, with amplitudes for weak and neutral tastes differing only in those who reported low hunger. As hunger was not controlled in this study (rather, appetite ratings were taken and groups were formed on the basis of a median split) and as this was an isolated effect amongst a great deal of data, it is difficult to make any specific inferences about this finding. However, it does fit with previous

reports of early ERP deflections being modulated by appetite (e.g., Stockburger et al., 2008; Plihal et al., 2001). The effects of hunger and satiety on taste processing are examined in detail in Chapter Four.

The source estimations indicate that the greatest activations at the early latency originated from the right POP in the PGC. This supports previous findings of early taste processing originating from this area (e.g., Mizoguchi et al., 2002; Ianilli et al., 2014; Ohla et al., 2010b), although no intensity dependent current density differences were found. In the later latency (1000 – 1500 ms), the greatest activations were observed in the right inferior parietal lobule, an area comprising both primary and secondary somatosensory regions (e.g., Penfield & Jasper, 1954). The same region was shown to be activated in studies investigating electric taste (e.g., Ohla et al., 2010), with stronger activations for greater taste intensities. We did not observe any intensity dependent differences in this region; however, given that the experience of taste requires a somatosensory component, it is not surprising that activations occurred here.

The early right-lateralised source activations differ from the left sided early ERP effects and during the later latency, the source activations were more posteriorly located. However, the ERP permutation method is designed to determine electrode clusters where differences between conditions emerge, and not necessarily the greatest activations. Our results suggest that the right POP is activated most during early taste processing, the right inferior parietal lobule is activated most during later latencies and that differences in taste intensity evoke a left-parietal and right frontal ERP effect.

An unexpected finding emerged within the taste intensity ERP data; in the left parietal region the neutral (water) and the medium intensity (0.1 M NaCl) tastants evoked equally decreased amplitudes at both early and late epochs, with the medium taste intensity evoking significantly different amplitude from the weak in the earliest epoch. This effect is curious, as we might expect similar processing to be more likely to occur for lower concentrations of salt that are perceptually and chemically more similar to water. However, the 0.1 M NaCl tastant represents a concentration that is similar to NaCl levels found naturally within saliva (0.08 M; Pfaffmann, 1959). On a cognitive level, it is possible that this similarity meant the 0.1 M NaCl concentration was not sufficiently arousing to induce a more

pronounced neural response and that preferential neural engagement occurred for the more distinct taste intensities. Alternatively, on a chemical level, the 0.1 M NaCl solution may not have caused sufficient change in the salivary environment to induce variations in taste fibre activity and consequent neural amplitude augmentations that differed from those following delivery of water. However, a problem with these interpretations is that all participants were able to correctly discern the different taste qualities, and rated 0.1 M NaCl as significantly more intense than 0.05 M NaCl. Furthermore, an analogous effect was not apparent in the ERD data, which instead showed a more predictable similarity in processing between neutral and weak and between medium and strong conditions. It may also be the case that the limited quantity of trials remaining (average 21.71), after cleaning and removing a significant amount of noise from the data, was not sufficient to detect changes in ERP processing that may have occurred (Mizoguchi et al., 2002). Further research examining taste ERP processing, with increased stimulus repeats, is required to fully understand these effects.

Within the ERD/S data, intensity was represented in alpha (7 – 13 Hz) and beta-band (13 – 30 Hz) oscillations in the central-parietal region around 2 s after stimulus onset. In each case, desynchronization of oscillations occurred in response to the medium and strong conditions, possibly due to the increased attention and arousal evoked by these stimuli (Klimesch et al., 2006; Palva & Palva, 2007; Thut et al., 2006; Worden et al., 2000). In contrast, neutral and weak tastes evoked little change, or moderate alpha-synchonization typically associated with decreased, or suppressed neural engagement (Klimesch et al., 2006; Pfurtscheller, 2003). Thus, unlike the early ERP data, the oscillatory findings present an uncomplicated picture of gustatory intensity processing, whereby the more perceptually similar intensities are processed analogously. Time-frequency representations may therefore provide the most useful method of assessing EEG responses to this characteristic of taste.

Taste quality

The effects of taste quality on ERP data were observed in later epochs (at 680 – 780 ms and 1200 – 1500 ms) in right parietal regions, where water evoked decreased amplitudes compared with other tastes. The general location of these effects conform somewhat with previous findings (e.g., Crouzet et al., 2015). However, unlike earlier reports our effects appeared relatively late. Such late-onset effects have generally

been linked with the distinction between neutral and arousing events (Hajcak et al., 2010b), so the present data may reflect greater neural resources being dedicated to arousing non-water tastes rather than specific taste qualities.

The source estimations suggest that the right POP and the right inferior parietal lobule were most activated during the early and late latencies respectively. These regions correspond with the ERP data, however, there were no differences between the current densities of the taste quality conditions. Crouzet et al. (2015) also reported activations in the opercula area for different taste qualities, although their reported activations began much earlier, they too reported no differences in activations for different taste qualities. Our findings further highlight the difficulties in determining distinct coding mechanisms for differing taste qualities.

The ERD/S data indicated right fronto-central theta-band, and left central-parietal alpha-band oscillations around 2 s were modulated by taste quality. All tastes qualities showed centralised theta-band ERD, which was greatest in response to bitter tastes; whereas alpha-band ERD was greatest for sweet tastes. Our data conform with those of Tóth and colleagues who found right-frontal theta ERD in response to the bitter taste of tea (Tóth et al., 2004). Theta-ERD is generally associated with inactivity, or a suppression of activity in areas not associated with the task (Kawamata et al., 2007). This suppression of activity could extend to habituation, as theta-band ERD has been found to occur in relation to the habituation of arousing or novel events (Irmiš et al., 1970; Kemp & Kaada, 1975; Sainsbury, 1970). Moreover, in the right amygdala - a theta-inducing region, BOLD signal decrement has been shown to occur in response to habituation to emotional events (Wright et al., 2001). Thus, the current results may reflect habituation, although it is unclear why greater habituation occurred in response to bitter stimulation.

Taste hedonicity

The effects on the ERPs of taste hedonicity were not detected until the later processing epoch (600 – 1500 ms) when we observed differential responses to neutral relative to hedonically positive and hedonically negative tastes in the right frontal regions. This late effect fits with current models of sensory and emotional processing which highlight later processing epochs dedicated to coding affective characteristics (e.g., Calvert, 2001; Dematte et al., 2008; Hajcak et al., 2010b; Ohla

et al., 2012). The similarity in amplitudes observed in response to pleasant and unpleasant tastes reinforce the previously reported difficulty in distinguishing between ERP responses to equally arousing but hedonically distinct stimuli (Hajcak et al., 2010b).

In contrast, the source localisation data were able to discern differences in the responses to different taste hedonicities. Estimations suggest that the greatest activations at the 600 – 1500 ms latency occurred in the right inferior parietal lobule, where activations were greatest for sweet tastes and weakest for bitter tastes. Similar results were reported in Zald et al. (2002), where greater activations for sucrose compared with QHCl and water were also found in this region. This region has previously been implicated in taste intensity processing (e.g., Ohla et al., 2010), however, these findings suggest a more affective role for the inferior parietal lobule in taste processing.

Our data also intimates that ERD may lend itself to the separation of the confounding influences of arousal and hedonic evaluation. Specifically, we were able to discern a distinct left lateralised alpha-band ERD response to pleasant tastes, differentiated from those evoked by either neutral or unpleasant stimuli. These findings fit well with evidence of left hemispheric alpha-specificity for pleasant events (Balconi & Mazza, 2009; Davidson & Henriques, 2000; Waldstein et al. 2000) and, importantly, extend this specificity to the processing of hedonically positive gustatory information. Similar responses, however, were not observed within the beta-band frequency. Instead, both hedonically positive and negative tastes produced similar left parietal-occipital beta-band ERD that differed reliably from the response to neutral stimuli. This finding is suggestive of a response that might be attributed to intensity or arousal rather than hedonicity.

Limitations

We recognise that we failed to produce distinctive peak ERP components (see Figure 3.3): rather, clusters of differential activity were observed. However, typical peak potentials in response to gustatory information, as indicated earlier, are difficult to obtain since they require repetitive stimulus presentations (Mizoguchi et al., 2002), balanced with long ISIs (Evans et al., 1993) and is not uncommon for gustatory ERP effects to be reported in the absence of peak components (Crouzet et al., 2015;

Prescott, 1994; Singh et al., 2011). In the current study, the extensive stimulus set and ISI had to be offset by limiting the number of repetitions within each stimulus category in order to provide a feasible testing duration. In addition to this, we experienced substantial noise within the data resulting from head movements. Participants reported feeling uncomfortable during the later trial blocks due to the length of the investigation and the volume of liquid ingested. This movement resulted in a significant loss of trials, which further limited the volume of data available for analysis. Our subsequent investigations have shown that increasing the quantity of stimulus repetitions and reducing testing durations can result in fully articulated gustatory ERP components (Chapter Five). Nevertheless, to our knowledge, this is the first human study to examine electroencephalographic responses to such a broad range of tastants, and to all intents and purposes, it was an ERP analysis that was performed. Given that the stimulus set was carefully designed to differentiate responses to taste quality, intensity and hedonicity, in a way that has not previously been attempted, the forfeiture of peak components must be weighed against the comprehensive nature of the stimuli employed. In the absence of typical ERPs, we were nonetheless able to establish specific patterns of activation in the processing of taste information originating from primary gustatory cortices. As noted earlier, however, the limited trial repeats may have affected the ability to discern some ERP amplitude changes that may have been observable had there been more available data.

Conclusions

We conclude that with the present methodology, scalp recordings of cortical electrical activity in humans reveal differential temporal, regional and oscillatory coding for specific taste characteristics. When applying a diverse stimulus set that could reliably differentiate between taste quality, intensity and hedonic value, we found evidence to suggest that, as previously observed in ERP data for other modalities, simple stimulus features such as intensity are processed early and affective attributes are coded later. Moreover, the latencies of the early ERP effects corresponded with activations in primary gustatory regions and later differences observed for taste hedonicity were associated with differential activity in regions previously associated with somatosensory processing. However, the observed patterns of activity did not fit precisely with the psychophysical attributes of the

tastes and indicated confounding influences of arousal. Similarly, ERP results for taste quality did not precisely distinguish between distinct tastes; rather, some of the effects indicate that more arousing tastes (i.e., sweet and bitter) receive more processing resources than less arousing taste qualities (i.e., water).

In contrast, the ERD/S data did show distinct differences in the coding of neutral/weak relative to medium/strong taste intensities, and both ERD/S and source-localisation analysis were able to differentiate between levels of taste hedonicity. These data fit well with current thinking on the involvement of neural oscillations in sensory and affective coding (e.g., Basar et al., 2012; Eckhorn et al., 1988; Engel et al., 2001; Kayser et al., 2012; Laurent & Davidowitz, 1994; Nicolelis et al.,1995) and, for the first time, demonstrate their relationship to the psychological evaluation of taste. Moreover, the data highlights that the inferior parietal lobule may play a role in coding taste hedonicity and this should be explored further.

However, the ERD and source-localisation data also failed to dissociate individual taste qualities. Rather, the results seem to imply habituation to bitter tastes as seen in increased right-lateralised theta ERD, and to the pleasantness of the sweet tastes, as indicated in the increased left-lateralised alpha ERD. These data further reflect the difficulty of determining distinct coding attributes for different taste qualities in what may be a broadly tuned mechanism (Schiffman, 2000; Smith & St John 1999) encompassing both spatial and temporal processing schemes (e.g., Crouzet et al., 2015). Therefore, taste quality coding may be better determined using more precise imaging methods such as intracranial recordings and high-density fMRI.

Overall, gustation involves complex mechanisms, beginning with transduction at receptors in the oral cavity and ending with representations of stimulus attributes in primary and secondary gustatory systems in the brain. The present study goes some way to characterise these representations, and suggests that analysis of neural oscillations may be the key to characterizing the coding of intensity and hedonic attributes of taste, although solutions to discerning the processing of taste quality remain to be elucidated.

Chapter Four: The Effects of Hunger and Satiety on the Processing of Gustatory Information.

4.1. Abstract

Physiological states of hunger and satiety can affect taste perception, but the neural underpinnings of this relationship have yet to be characterised. In this chapter we explored the effects of hunger and satiety on the neural processing of pleasant (sweet), unpleasant (bitter) and neutral (water) tastes. Sixteen healthy adults were tested on two occasions after a 12-hour overnight fast, either when hungry or when sated after consuming a liquid meal. EEG was recorded relative to the onset of the tastant and ERPs, ERP source-localisation and ERD/S were examined. Behavioural data revealed that sweet tastes were rated as less pleasant when sated. Taste responses were observed in limbic regions under both hungry and sated conditions and different tastes were distinguished in early (90 – 160 ms) and late (500 – 1500 ms) ERP epochs and within theta-, alpha- and beta-band oscillations. Hunger enhanced ERP and beta-ERS responses overall, however, sweet taste responses were dependent on hunger state and showed increased ERP and alpha-ERD under sated conditions. The data suggests that hunger state modulates the processing of tastes with differential attentional and evaluative processes employed under hungry and sated conditions, particularly for sweet tastes.

4.2. Introduction

Appetite and taste perception are crucial mechanisms that work together to guide eating behaviours. The gustatory properties of food not only play an important role in the identification and selection of edible substances (e.g., Blundell et al., 2010; Scott et al., 1995), but taste-cued reflexes also facilitate processes that are necessary for digestion to take place (e.g., Katschinski, 2000). The role of taste in appetite has been well explored and findings suggest taste perception has an acute affect on hunger and eating behaviours (Cerf-Ducastel & Murphy, 2003; Ferdon & Murphy, 2003; Fukunaga et al., 2005; Hays & Roberts, 2006; Murphy & Gilmore, 1989; Murphy et al., 2002; Rolls & McDermott, 1991; Schiffman et al., 1979; Yeomans, 2000; Yeomans, 1996; Yeomans & Symes, 1999). Less explored, however, is the reverse effect of the influence of appetite in the perception of tastes. Nevertheless, evidence is emerging that physiological states of hunger and satiety may be influencing the perception of taste and that this effect can be observed at a neurological level (Del Parigi et al., 2004; Gautier et al., 2000; Gottfried et al., 2003; Hasse et al., 2009; Kringelbach et al., 2003; Tataranni et al., 1999; Uher et al., 1996).

Anecdotally, people often report that food 'tastes better when you are hungry' (e.g., Zverev, 2004), and it has long been posited that hunger and satiety influence taste preferences (e.g., Albanese, 1957; Albanese et al., 1955; Mayer - Gross & Walker, 1946; Pangborn, 1959). However, the evidence for this effect has been somewhat mixed. In an early study of 11,456 consumers at a state fair, Pangborn (1959) reported that appetite was unrelated to the pleasantness ratings of canned peaches. However, a later investigation found that sweet solutions that are rated as palatable when an individual is hungry become less pleasant after ingestion of glucose syrup; in a process termed 'Alliesthesia' (Cabanac, 1971, see also Fantino, 1984, for a review).

Similarly, Rolls et al. (1981) found that after consuming a particular food to satiety, the palatability of that food declined relative to other, non-consumed foods (sensory-specific satiety; e.g., Hetherington, et al., 1989; Rolls, et al., 1981; Rolls, et al., 1983; Rolls, 1985). Sensory-specific satiety effects have been demonstrated by studies asking participants to rate their subjective experience of tastes or foods following the consumption of a meal (e.g., Rolls et al., 1981). The most consistent finding of these investigations is that eating a savoury (salty) meal to satiety leads to

a decrease in the pleasantness of that food and of other savoury foods. However, the same phenomenon has been reported for eating a sweet food to satiety (e.g., Rolls et al., 1984). This process is considered to be part of a mechanism that regulates short-term food intake.

Other studies have reported that it is taste discrimination processes, rather than hedonic processes that are affected during states of hunger and satiety. Moskowitz et al. (1976) found that under fasting conditions, pleasantness ratings for increasing concentrations of glucose reached a maximum point then decreased. Conversely, under sated conditions, the pleasantness ratings for the glucose solutions continued to increase with concentration. The authors concluded that following satiation; individuals are no longer able to discriminate the palatability and intensity of sweet taste solutions. However, although recorded, the authors did not report ratings for unpleasant tastes, so it is unclear whether diminished discrimination of taste affect and intensity following satiation appears across all tastes, or whether it is specific to palatable or nutrient rich tastants.

In addition to palatability, it has been suggested that hunger and satiety may modulate taste intensity perception (Glokner, Fikentsher & Ulrich, 1986; Kawai et al., 2000; Shigemura et al., 2004; Zverev, 2004) although, again, the evidence is mixed (e.g., Pasquet et al., 2006). Zverev (2004) found that that sensitivity to NaCl and sucrose was increased during fasted, compared with sated states, whereas Pasquet et al. (2006) and Pangborn (1956) reported no differences in taste sensitivity between hungry and sated participants.

While behavioural findings are mixed, some interesting neural data has been obtained (Del Parigi et al., 2004; Gautier et al., 2000; Gottfried et al., 2003; Hasse et al, 2009; Kringelbach et al., 2003; Tataranni et al., 1999; Uher et al., 1996). As discussed in Chapter One (section 1.7.2), in response to food stimuli, consistently greater activations within the insula and thalamus under hungry conditions have been observed, with increases in OFC activations found under sated conditions (Del Paragi et al., 2002; Hasse et al., 2009; Tataranni et al., 1999; Uher et al., 1996).

In a recent fMRI investigation, Hasse et al. (2009) reported that both pleasant (e.g., sucrose) and unpleasant (e.g., citric acid) tastes evoked increased insula activations when hungry, with greater overall global activations reported for sucrose. In the sated condition, all tastes evoked decreased activations in limbic

areas involved in motivation and reward. These results suggest that the state of hunger elicits activations from regions involved in the sensory processing of tastes and satiety deactivates regions associated with motivation and reward, perhaps as a mechanism to terminate food intake. This process may be mediated by the meal used to induce satiety. For instance, contrary to previous findings (e.g., Del Paragi et al., 2002), OFC activations in response to a liquid food recently consumed to satiety have been shown to decrease in line with reductions in pleasantness ratings (Kringlebach et al., 2003), and activations in both taste and reward areas have been shown to decline in response to oral fat, following a high fat meal (compared with a water load: Eldeghaidy et al., 2016).

In our own investigations, we found indirect evidence to suggest that hunger may modulate ERP responses to tastants (Chapter Three), with weak and neutral taste intensities only discriminated in neural responses when participants reported reduced hunger. Other studies have reported increased ERP amplitudes for food stimuli in hungry states (e.g., Stockburger et al., 2008), but not for stimuli unrelated to food (e.g., Gesiler & Polich, 1990; 1992). Only one EEG study has examined the affect of hunger on taste processing directly. In that study, Jacquin-Piques et al. (2016) measured ERP responses to repeated stimulation of a sucrose concentration in participants under hungry and sated conditions. The authors reported little effects of appetite on taste processing, only that gERP latencies from frontal electrodes (reported as recording PGC activity) lengthened when sated, with no changes in amplitude observed. The lack of effects here is surprising, given the reported differences observed in fMRI studies (e.g., Del Paragi et al., 2002; Hasse et al., 2009; Tataranni et al., 1999; Uher et al., 1996) and may due to the appetite manipulations employed (see Chapter One, section 1.7.2).

In this chapter, we aimed to explore the effects of hunger and satiety on the behavioural and electrophysiological processing of tastes. Understanding the temporal mechanisms of the hunger-taste relationship could provide important insights into the processing stage at which hunger is relevant to taste encoding and the oscillatory mechanisms involved in communicating this information. We explore the processing of both pleasant and unpleasant tastes to determine if the modulation of taste responses by appetite is specific to palatable tastes, or is a general mechanism for all tastants. We also employed stringent controls of hunger and

satiety by testing participants after a 12-hour fast, and following a liquid meal constituting half of each participant's daily resting state energy requirement, and confirm fasting and fed states by measuring plasma blood glucose levels (e.g., Tataranni et al., 1999). Based on the findings from Chapter Three, we reduced the stimuli set to three tastants (sucrose, QHCl and water) and increased repetitions in order to reduce study duration and noise components; to attempt to provide more articulated ERP components. We also added an arousal measure to try to determine if any ERP effects can be attributed to the arousing nature of the stimuli.

4.3. Materials and Methods

4.3.1. Participants

Due to recorded gender and BMI differences in neural responses to hunger and satiety (e.g., DelParigi, 2002; Gautier, 2000; Wang, 2009), only female participants were employed, with obese (BMI > 30) and under-weight (BMI < 18.5) individuals excluded from participation. Sixteen normal- to marginally over-weight (BMI range, 19.9 – 25.6; M = 23, SD = 1.8) female participants aged 20 – 31 years (M = 26.13, SD = 4.0) took part in the EEG study[1]. All participants were pre-screened and were non-smokers, non-diabetic, had no food allergies or intolerances or taste disorders, and were not taking medications or suffering illnesses that interfered with their gustatory, olfactory or hunger perception or would prevent a 12-hour fast. All participants gave informed consent and the study was conducted in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki), and was approved by the University of Liverpool Ethics Committee.

4.3.2. Design

A 2 × 3 within-subjects design was employed. The independent variables were taste (bitter, sweet, water) and hunger state (hungry, sated). The dependent variables were ERP amplitude, current densities at ERP latencies and ERD/S power.

4.3.3. Taste stimuli

The selection of taste stimuli is described in Chapter Two (section 2.3). We selected a concentration of bitter quinine HCl (0.0003 M) and sweet sucrose (0.3 M) for their

[1] Variations in BMI had no effect on behavioural or EEG data (ps > .168).

discernibly different ratings of pleasantness, but similarly rated intensity. Water was selected as neutral control, differing in both intensity and pleasantness from the bitter and the sweet solutions (*p*s < .019).

4.3.4. Measurements

Appetite was measured using a six part VAS scale (0 – 100) measuring hunger, fullness, desire to eat, satisfaction, nausea and thirst (Flint et al., 2000; Rolls et al., 1999). To corroborate fasting and fed requirements, blood glucose concentrations were analyzed using finger-prick samples obtained with a Precision Xtra (Abbot Diagnostics, USA) amperometric glucose monitor, which was calibrated to give plasma-equivalent glucose results. During testing, each taste was rated for pleasantness and intensity using the LAM (Schutz & Cardello, 2001) and gLMS (Bartoshuk et al., 2004) respectively. Arousal was measured using a vertical VAS scale (0 = not at all arousing, 100 = extremely arousing). As summarised in Table 4.1, the provision of the liquid meal was effective in reducing appetite levels, with paired sampled t-tests revealing significant differences between the hungry and sated conditions for each measure. There was also a reliable rise in post-meal blood glucose in the fed compared with the fasting condition. Additionally, participants reported lower levels of thirst and increased nausea in the fed condition

Taster status was also recorded but had no effect on the behavioural or EEG findings (*p*s > .132) so this factor is not discussed further.

Table 4.1.

Measure	Hungry	Sated	*t* (*df* = 15)	*p*
Hunger	74.75 (19.2)	3.81 (5.14)	15.07	<.001 ***
Fullness	10.75 (12.76)	86.56 (12.80)	19.67	<.001 ***
Desire to eat	64.31 (26.57)	6.56 (9.02)	9.22	<.001 ***
How much could you eat	71.31 (21.92)	12.5 (15.93)	11.56	<.001 ***
Thirst	59.0 (18.82)	32.94 (24.95)	3.68	.002 **
Satisfaction	25.56 (18.19)	80.63 (12.34)	9.76	.001 **
Nausea	16.13 (19.7)	44.69 (31.63)	3.94	<.001 ***
Plasma glucose concentration	4.95 (0.50)	6.49 (0.45)	9.57	<.001 ***

Table 4.1 The mean (± SD) ratings for each measure in the appetite scale (measured with 100 mm VAS) and the mean (± SD) plasma glucose concentrations (mg/dL) in both hungry and sated conditions. *T* tests compared mean ratings for each measure and results are reported in the table. Asterisks indicate significant differences between conditions assessed by paired *t*-tests: ** $p < .01$ *** $p < .001$.

4.3.5. Stimulus presentation

The taste stimuli were presented using the same gustometer method as described in Chapter Three. The order of the taste samples was pseudorandomised, with steps taken to ensure that the same taste sample was never presented on two consecutive trials. In this study, the tastants were followed by ratings scales to measure the pleasantness, intensity and arousal of the tastes. Figure 4.1 shows a schematic of the stimulus presentation procedure. Overall, each of the three stimuli was repeated 40 times over 120 trials, separated into 4 blocks of 30 trials.

4.3.6. Procedure

All participants were tested on two separate occasions (one week apart), each after an overnight fast. Participants were tested between 08:30 and 09:30 and were instructed not to consume any food or beverage other than water for 12 hours prior to each session. In the sated condition, participants were required to consume a liquid meal (Ensure Plus, 1.5 kcal/ml, Ross-Abbot Laboratories, Columbus, OH), accounting for 50% of their daily resting energy requirement, determined using the basal metabolic rate calculator (Schofield, 1985; mean energy intake = 732.62 kcal, *SD* = 57.5). After 20 minutes, participants completed the appetite questionnaire and their blood glucose level was measured. The EEG equipment was fitted to the participant who was then seated in the experimental chamber. After completing a practice trial, the main experiment began with stimuli delivered and LAM, gLMS and arousal rating scales as described above. The fasting condition followed the same procedure but without the liquid meal prior to the glucose measurement. The order of the sated and fasting conditions was randomised. Overall, the experiment took 1 – 1.5 hours hour to complete.

4.3.7. Electrophysiological measures: ERP and ERD/S

The ERP, sLORETA and ERD/S data were recorded, cleaned and pre-processed using the same methods described in Chapter Three. An average of 8.8 (*SD* = 3.5) noise components were removed using ICA. No participant incurred a loss of > 50% of trials in any one condition, thus no participant data were removed from EEG analyses. There was an average (mean ± *SD*) of 24.56 (± 5.57) sweet taste trials remaining, 26.7 (± 4.11) bitter trials retained and 31.38 (± 5.98) water trials remaining for each participant following pre-processing procedures. Significantly

more trials were retained for water than for bitter tastes (MD = -6.19, SE = 1.07, p < .001).

4.3.8. Statistical analysis

As with Chapter Three, we performed three types of analysis: the standard time-domain averaging technique to examine ERPs, sLORETA to examine the origin of the ERP effect and a wavelet-based TFR to analyse underlying neural oscillations in the form of ERD/S. The statistical analysis procedures for each of the EEG analyses were the same as described in Chapter Three. Once identified, the ERP clusters, sLORETAs and ERD/S clusters were evaluated for each participant, and subjected to a series of within-subjects ANOVAs with the factors: taste (bitter, sweet, water), hunger (hungry, sated) and taste × hunger state.

Post hoc analyses using pairwise comparisons and Bonferroni corrections were conducted for each EEG analysis when significant effects occurred. Greenhouse-Geisser corrections were applied when statistical assumptions were not met. Where multiple significant effects occurred, results were collated to show the smallest mean difference, greatest standard error and greatest p values respectively (MDs >, SEs <, ps <). Effect sizes (ES) represent the partial η^2 value.

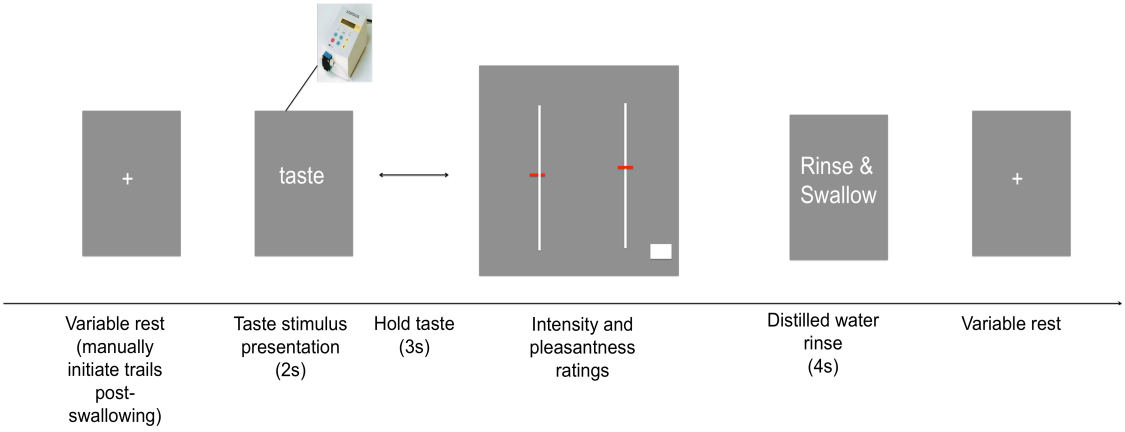

Fig. 4.1. A schematic diagram of the experimental procedure**.**

4.4. Results

4.4.1. Behavioural analysis

Intensity ratings

We examined the affects of hunger state (hungry, sated) and taste (bitter, sweet water) on taste intensity ratings gathered over the course of the experiment [Figure 4.2 (a)] using a 2 × 3 repeated measures ANOVA.

There was a main effect of taste on intensity ratings, F (2, 30) = 84.77, p < .001, ES = .85, with all tastes rated differently from each other (*MDs* > 13.76, *SEs* < 4.31, p < .003). Bitter was rated as the most intense, followed by sweet and then water [Figure 4.2 (a)]. There was no effect of hunger on taste intensity (p = .816) and no interaction between hunger and taste on intensity ratings (p = .215).

Pleasantness ratings

As with the intensity ratings, we examined the influence of hunger state (hungry, sated) and taste (bitter, sweet, water) on taste pleasantness ratings gathered over the course of the experiment [Figure 4.2 (b)] using a 2 × 3 repeated measures ANOVA.

The three taste solutions were reliably distinguished by their pleasantness ratings, F (2, 30) = 64.93, p < .001, ES = .81; *MDs* > 13.76, *SEs* < 4.31, p < .003, with sweet rated as pleasant, bitter as unpleasant, and water rated neutrally. There was also a significant interaction between hunger state and taste, F (2, 30) = 9.85, p < .001, ES = .40, with sweet taste being rated as less pleasant when participants were sated (M = - 6.42, SE = 2.74) than when they were hungry (M = 16.27 SE = 2.74). There was no influence of hunger state on pleasantness ratings of bitter or water.

Arousal ratings

We also examined the effect of hunger state and taste on taste arousal ratings gathered over the course of the experiment [Figure 4.2 (c)] using a 2 × 3 repeated measures ANOVA. There was a significant effect of taste on arousal ratings, F (2, 30) = 35.20, p < .001, ES = .70, whereby all tastes differed significantly from each other (*MDs* > 10.52, *SEs* < 6.19, p < .001), with bitter rated as the most arousing, followed by sweet and then water. There was no effect of hunger state on taste arousal ratings (p = .816) and no interaction between hunger state and taste on

arousal ratings (p = .350). Arousal measures showed a strong positive correlation with intensity ratings (r = .99).

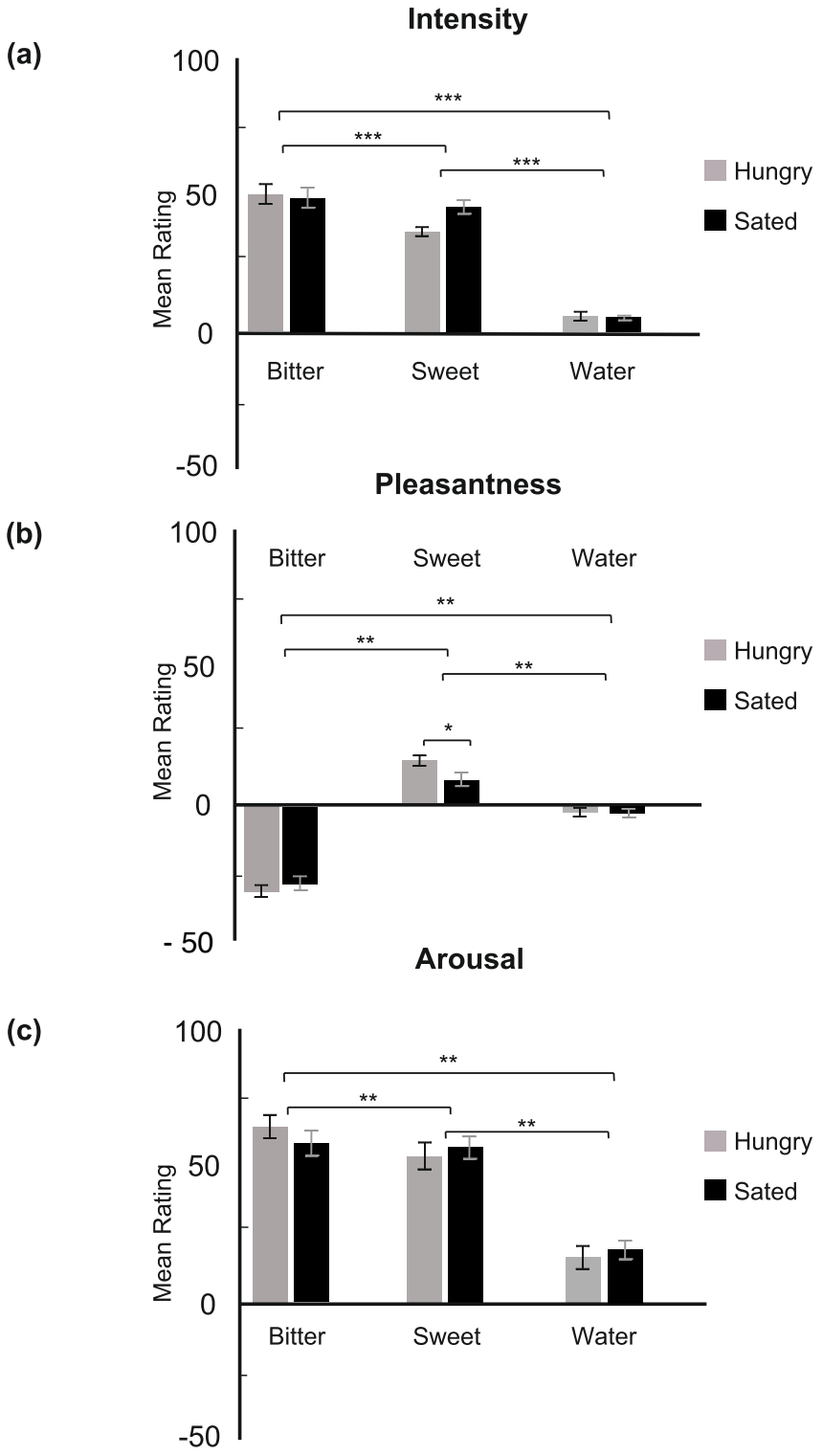

Fig. 4.2. Bar charts representing the mean (a) intensity, (b) pleasantness and (c) arousal ratings for each taste under hungry and sated conditions. Error bars indicate standard error. Asterisks indicate significant differences: * $p < .05$; ** $p < .01$, *** $p < .001$.

4.4.2. ERP analysis

As Figure 4.3 illustrates, no fully articulated ERP waveform could be observed. However, distinct temporal and regional differences in ERP magnitude were apparent for taste, hunger and hunger × taste interactions (Figure 4.4). The principal effects are summarised below for each factor.

Taste

Our analysis revealed significant effects of taste on EEG amplitude in the left frontal region (Af3, F1, F3, F5, F7, Fc5, Fc3, Fc1, C1, C3, C5) between 90 – 160 ms, F (2, 30) = 7.36, p = .003, ES = .33. From the ERP plot and topographic maps [Figure 4.4 (a, b)], we can see that compared with water, which showed little change at any point, both bitter and sweet tastes tended to evoke negative amplitudes. Bitter evoked significantly greater negative amplitude than water (*MD* = 0 .73, *SE* = 0 .23, p < .001) but did not differ from sweet (p = .103). The difference between sweet and water responses was not significant (p = .084). More marked effects were also apparent in the same region between 800 – 1500 ms, F (2, 30) = 12.96, p < .001, ES = .46. Again, there was greater negative amplitude evoked by bitter, but here both bitter and sweet responses differed reliably from water (*MDs* > 0.98, *SE*s < 0 .32, ps < .008). There were no significant differences between responses to bitter and sweet tastes over this interval (p = .106).

An effect of taste on EEG amplitude also emerged in the left central-parietal region (Cp3, Cp1, P1, P7, P9, Po7, Po3) between 500 – 1500 ms, F (2, 30) = 8.39, p = .003, ES = .36, with both bitter and sweet evoking similar positive amplitudes (p = .462) that differed from water (*MDs* > 1.05, *SEs* < 0 .27, p < .02), which itself showed little change [Figure 4.4 (b, c)].

Hunger state

A significant effect of hunger state on ERP amplitude was detected at the left frontal region (Fp1, Af3, Af7, F1, F3, F5, F7) between 200 – 600 ms, F (1, 15) = 5.78, p = .03, ES = .28. As illustrated in Figure 4.4 (d, e), fasting evoked greater negative amplitude (*M* = -1.6, *SE* = 0.27) over this epoch compared with the satiated condition (*M* = -0.71, *SE* = 0.16). This effect was no longer apparent between 1000 – 1500 ms (p = .065).

A marked and sustained effect of hunger across the entire epoch (100 – 1500 ms) also emerged in the left central-parietal region (Tp7, Cp1, Cp3, Cp5, P1, P3, P5; F (1, 15) = 12.14, $p < .001$, ES = .45), with a much greater positive amplitude in the hungry condition (M = 2.12, SE = 0.51) than in the satiated condition, which showed little change [M = -0.28, SE = 0.10; Figure 4.4 (e, f)].

Taste × hunger state

There was a significant interaction between taste and hunger state on EEG amplitude in the left frontal region (F5, Fc5, FT7) between 1000 – 1500 ms, F (2, 30) = 4.67, p = .017, ES = .44. As may be seen from Figure 4.4 (g, h), this interaction was primarily generated by the differences between responses to sweet taste and water. Sweet taste evoked negative amplitude when sated (M = -2.39, SE = 0 .74) but not when hungry (M = 0.49, SE = 0.90). Conversely, water evoked greater amplitude when hungry (M = 0.92, SE = 0.56) compared to the more neutral response when sated (M = -0.36, SE = 0.47). By contrast, bitter evoked similar negative amplitudes when hungry (M = -1.50, SE = .59) and when sated (M = -1.58, SE = .50).

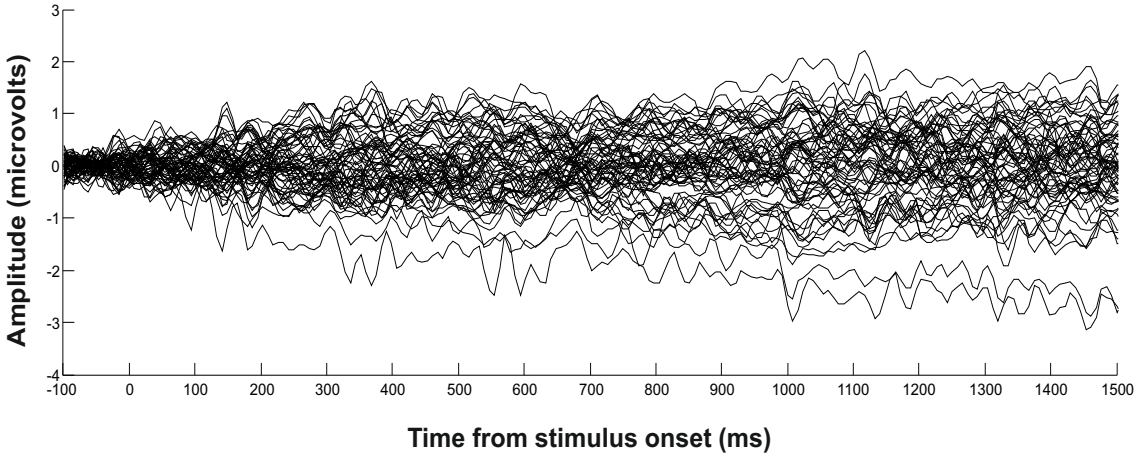

Fig. 4.3. Butterfly plot demonstrating the average ERP waveform across conditions for each electrode (separate lines) from the onset of the tastant.

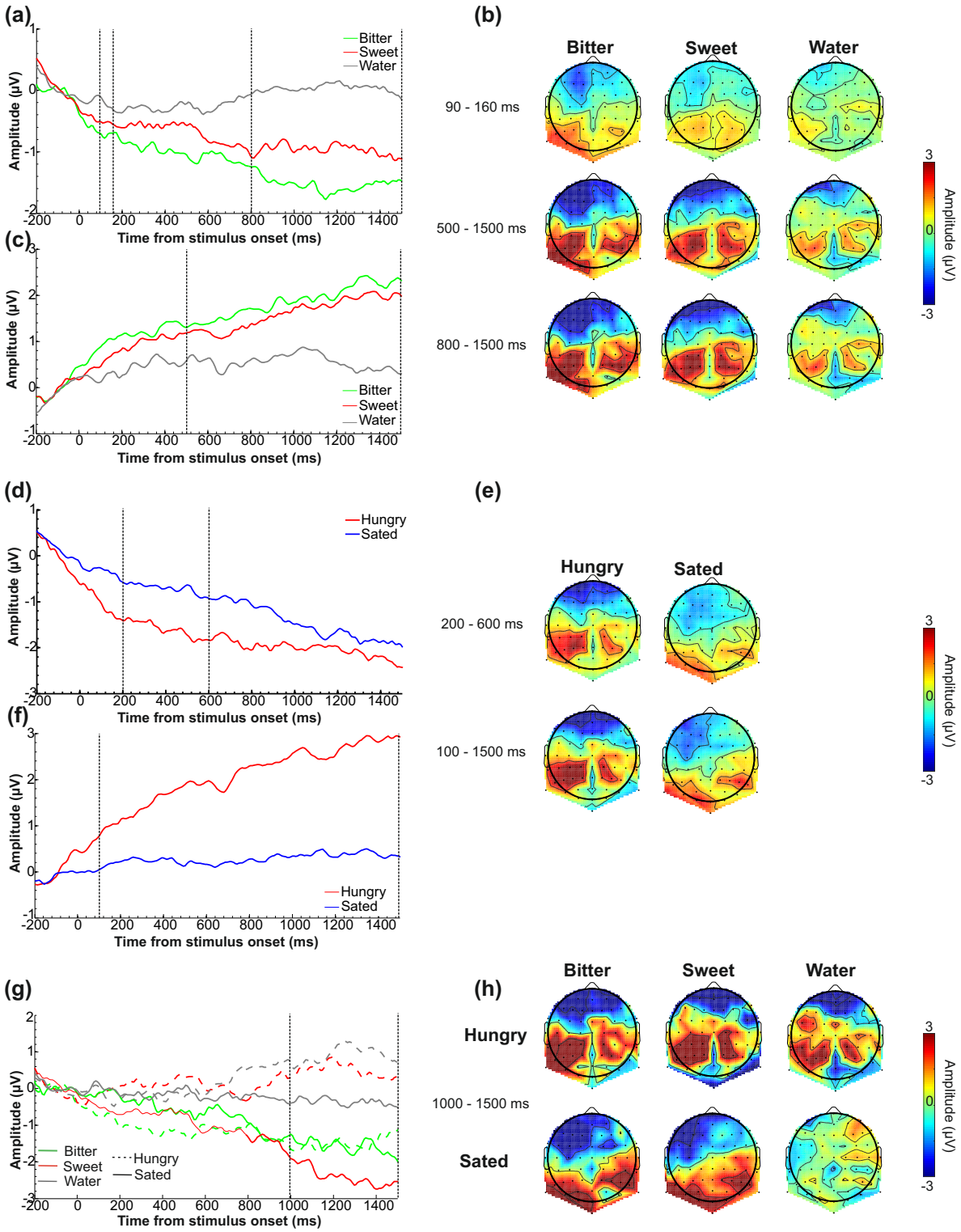
(a)
Bitter
Sweet
Water
Amplitude (μV)
Time from stimulus onset (ms)
(b)
Bitter
Sweet
Water
90 - 160 ms
500 - 1500 ms
800 - 1500 ms
Amplitude (μV)
(c)
Amplitude (μV)
Bitter
Sweet
Water
Time from stimulus onset (ms)
(d)
Hungry
Sated
Amplitude (μV)
Time from stimulus onset (ms)
(e)
Hungry
Sated
200 - 600 ms
100 - 1500 ms
Amplitude (μV)
(f)
Amplitude (μV)
Hungry
Sated
Time from stimulus onset (ms)
(g)
Amplitude (μV)
Bitter
Sweet
Water
Hungry
Sated
Time from stimulus onset (ms)
(h)
Bitter
Sweet
Water
Hungry
1000 - 1500 ms
Sated
Amplitude (μV)

Fig. 4.4. Temporal ERP amplitude plots with vertical dashed lines indicating intervals with significant main effects of condition, and topographic head plots with colour bars representing amplitude (μV). (a) Significant ERP effects of taste in the left frontal region (Af3, F1, F3, F5, F7, Fc5, Fc3, Fc1, C1, C3, C5) between 90 - 160 ms and 800 - 1500 ms. (b) Topographic headplots indicating taste effects between 90 – 160 ms, 500 – 1500 ms and 800 – 1500 ms. (c) Significant ERP amplitude effects of taste in the left central-parietal region (Cp3, Cp1, P1, P7, P9, Po7, Po3) between 500 – 1500 ms. (d) Significant ERP amplitude effects of hunger and satiety in the left frontal region (Fp1, Af3, Af7, F1, F3, F5, F7) between 200 - 600 ms. (e) Topographic headplots indicating hunger state effects between 200 -600 ms and 100 – 1500 ms. (f) Significant ERP effects of hunger in the left central-parietal region (Tp7, Cp1, Cp3, Cp5, P1, P3, P5) between 100 – 1500 ms. (g) Significant ERP effects of hunger state × taste interactions in the left frontal-temporal region (F5, Fc5, FT7) between 1000 – 1500ms. (h) Topographic headplots indicating taste × hunger state interaction effects between 1000 – 1500 ms.

4.4.3. sLORETA analysis

Figure 4.5 illustrates the sLORETA source estimates of each of the ERP latencies and the current densities for each condition. The principal sources are summarised below for each factor.

Taste

During the early taste latency (90 – 160 ms), activations were observed in the left cingulate gyrus [TAL, $x = 0$, $y = -34$, $z = 25$; Figure 4.5 (a)]. There was a significant effect of taste on current density, F (2, 30) = 10.14, $p < .001$, ES= .40, which was significantly increased in response to bitter tastes compared with the sweet and water [*MD*s > .27, *SE*s < .86, *p*s < .017; Figure 4.5 (b)]. During the 800 – 1500 ms latency, activations were observed in the left posterior cingulate gyrus [TAL, $x = -4$, $y = -32$, $z = 25$; Figure 4.5 (c)]. There was a significant effect of taste, F (2, 30) = 4.77, p = .016, ES = .24, whereby bitter tastes evoked a similar current density to sweet tastes (p = .154) but differed from water [MD = 0.24, SE = 0.07, p = .013; Figure 4.5 (d)]. During the 500 – 1500 ms latency, the greatest activations were again observed in the left posterior cingulate gyrus [TAL, $x = -4$, $y = -32$, $z = 25$; Figure 4.5 (e)]. There was a significant effect of taste, F (2, 30) = 3.72, p = .036, ES = .20, where again bitter tastes evoked a similar current density to sweet tastes (p = .19) but differed from water [MD = 0.17, SE = 0.05, p = .018; Figure 4.5 (f)]. However, when applying Bonferroni corrections, this effect fails to reach significance ($p < .017$).

Hunger state

During the 200 – 600 ms hunger state latency, activations were observed in the left cingulate gyrus [TAL, $x = 0$, $y = -34$, $z = 25$; Figure 4.5 (g)] but there were no effects of hunger state [p = .85; Figure 4.5 (h)]. During the long latency (100 – 1500 ms), activations were observed in the right posterior cingulate gyrus [TAL, $x = 5$, $y = -29$, $z = 25$; Figure 4.5 (i)], but no effects of hunger state on current density were observed [Figure 4.5 (j)].

Taste × hunger state

For the period when taste × hunger state interactions were evident in the ERP data (1000 – 1500 ms), the greatest activations were observed in the right middle frontal

gyrus [TAL, x = 53, y = -13, z = 34; Figure 4.5 (k)]. However, there was no significant interaction between taste and hunger state on the current density from this region [p = .618; Figure 4.5 (l)].

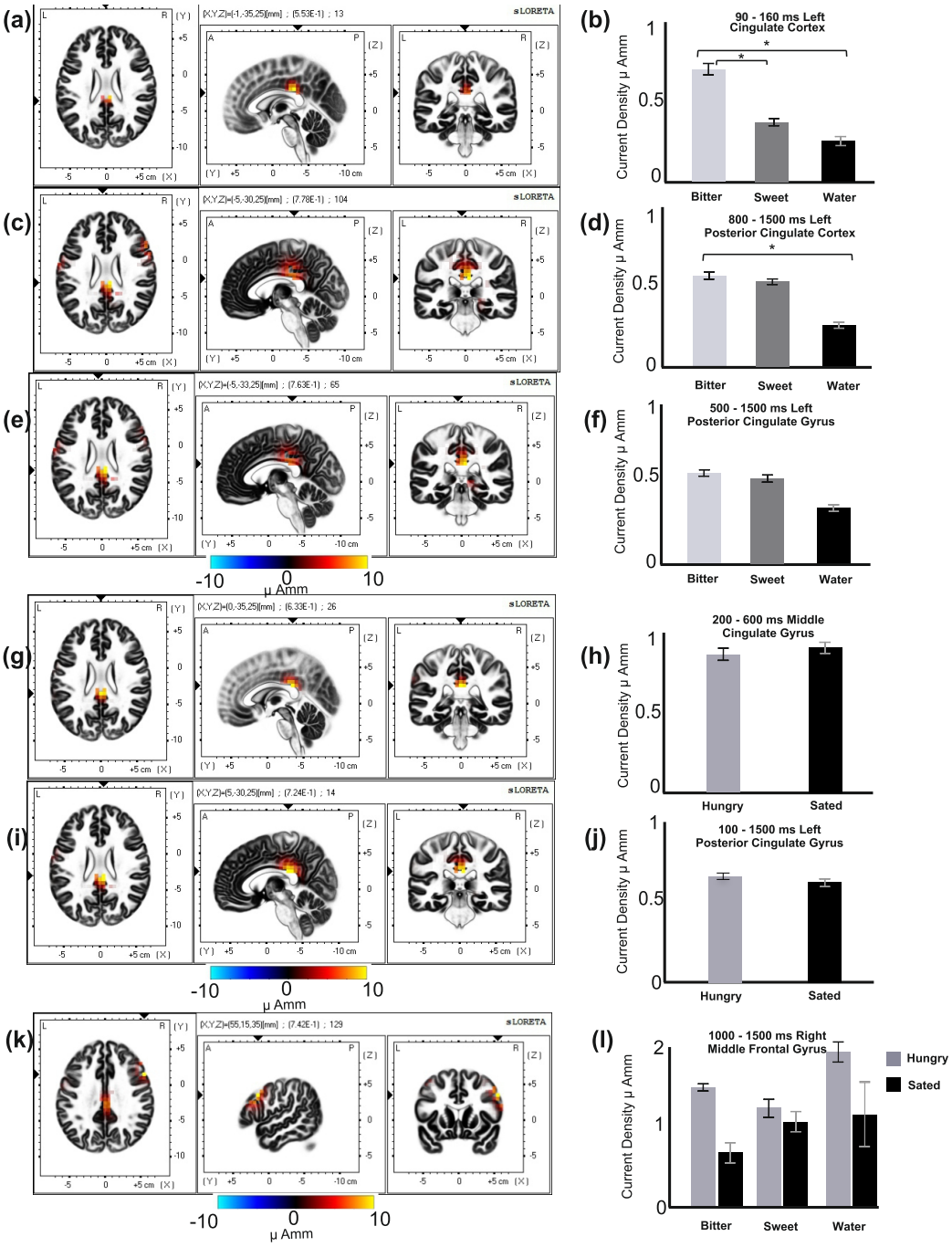
(a)
(b)
90 - 160 ms Left
Cingulate Cortex
Current Density μ Amm
Bitter
Sweet
Water
(c)
(d)
800 - 1500 ms Left
Posterior Cingulate Cortex
(e)
(f)
500 - 1500 ms Left
Posterior Cingulate Gyrus
-10
0
10
μ Amm
(g)
(h)
200 - 600 ms Middle
Cingulate Gyrus
Hungry
Sated
(i)
(j)
100 - 1500 ms Left
Posterior Cingulate Gyrus
(k)
(l)
1000 - 1500 ms Right
Middle Frontal Gyrus
Hungry
Sated

Fig. 4.5. sLORETA imaging results displaying maximum current density at each ERP latency for the grand mean results, with colour bars representing current density (μ Amm) and bar charts showing the current density for each condition, with error bars indicating standard error. (a) sLORETA image showing the maximum current density at 90 – 160 ms located in the left cingulate cortex. (b) Bar chart showing the mean current density at this location and latency for each taste condition. (c) sLORETA image showing the maximum current density between 800 – 1500 ms located in the left posterior cingulate gyrus. (d) Bar chart showing the mean current density at this location and latency for each taste condition. (e) sLORETA image showing the maximum current density between 500 – 1500 ms located in the left posterior cingulate gyrus. (f) Bar chart showing the mean current density at this location and latency for each taste condition. (g) sLORETA image showing the maximum current density between 200 – 600 ms located in the middle cingulate gyrus. (h) Bar chart showing the mean current density at this location and latency for each hunger state condition. (i) sLORETA image showing the maximum current density between 200 – 600 ms located in the middle cingulate cortex. (j) Bar chart showing the mean current density at this location and latency for each taste and hunger state condition. Asterisks indicates significance level: * $p < .05$.

Note – the sLORETA images show the MNI coordinates for each effect, these have been converted to Taliarach coordinates in the results section.

4.4.4. ERD/S analysis

As summarised in the time-frequency spectrographs and topographic maps in Figure 4.6, distinct differences in ERD/S were apparent for taste, hunger and hunger × taste interactions. The principal effects are summarised below for each factor in relation to theta-, alpha- and beta-band oscillations.

Taste

Analysis revealed significant effects of taste on theta-band oscillations (4 – 7 Hz) between 0.5 – 1.5 s. From Figure 4.6 (a, b), we can see that all tastes induced centralised theta-band ERD. Differences were apparent in the fronto-central region (Fp1, Fpz, Fp2, Afz, Fcz), F (2, 30) = 9.2, p = .001, ES = .38, whereby bitter and sweet tastes evoked decreased theta-band ERD compared to water, (*MD*s > 0.07, *SE*s < 0.33, *p*s < .03).

A main effect of taste on alpha-band oscillations (7 – 13 Hz) was also evident [Figure 4.6 (a, b)]. All tastes showed alpha-band ERS in the central parietal region, although this effect did not reach significance (p = .063). Nevertheless, reliable differences emerged in the right-frontal region (Afz, Af4, Fz, F2, F4, F6, Fc2, Fc4) between 2.0 – 2.5 s, F (2, 30) = 8.39, p = .001, ES = .36, where both water and sweet showed greater alpha-band ERD than bitter (*MD*s > 0.09, *SE*s < 0.04, *p*s < .013), which showed little alpha-band ERS.

An effect of taste on upper beta-band oscillations (20 – 30 Hz) also emerged across the bilateral frontal region (Fp1, Af3, F1, Af8, F2), F (2, 30) = 4.14, p = .026, ES = .22 [Figure 4.6 (a, b)]. Here, all tastes evoked slight beta-band ERS, but bitter taste elicited the greatest ERS and differed significantly from the responses to sweet and water (*MD*s > 0.07, *SE*s < 0.02, *p*s < .033). However, this effect disappears when applying Bonferroni corrections (p < .017).

Hunger

A significant effect of hunger on beta-band oscillations (13 – 30 Hz) was observed between 1.5 – 2.5 s across bilateral frontal-regions (F5, F7, Fc5, Fc3, Af8, Afz, F6, Fc6), F (1, 15) = 8.29, p = .011, ES = .36, and bilateral parietal regions (P2, P3, P4, P5), F (1, 15) = 8.29, p = .011, ES = .36 [Figure 4.6 (c, d)]. Specifically, significant beta-band ERS occurred in the hungry condition compared with minimal change of

activity in the sated condition (*MDs* > 0.06, *SE*s < 0.04, *p*s < .027). However, this effect failed to reach significance when applying Bonferroni corrections ($p < .017$)

Taste × hunger state

From Figure 4.6 (e, f), it is evident that there are differences between taste responses in the two conditions. Responses to bitter when hungry showed right-parietal alpha-band ERS, but were left-lateralised when satiated. In contrast, when hungry, left-parietal alpha-band ERS emerged in response to sweet taste, but when sated right-parietal alpha-band ERD was evident. There appeared to be little difference between conditions in the response to water. Statistically, there was a significant interaction between taste and hunger on alpha-band oscillations in the left central-temporal area (C1, C3, Tp7), between 1.5 – 2.5 s, generated by differential responses to sweet taste, F (2, 30) = 6.64, p = .009, ES = .15. When sated, sweet taste evoked alpha-band ERD (M = -0.09, SE = 0.03), compared to an ERS response when hungry (M = 0.07, *SE*s < 0 .04).

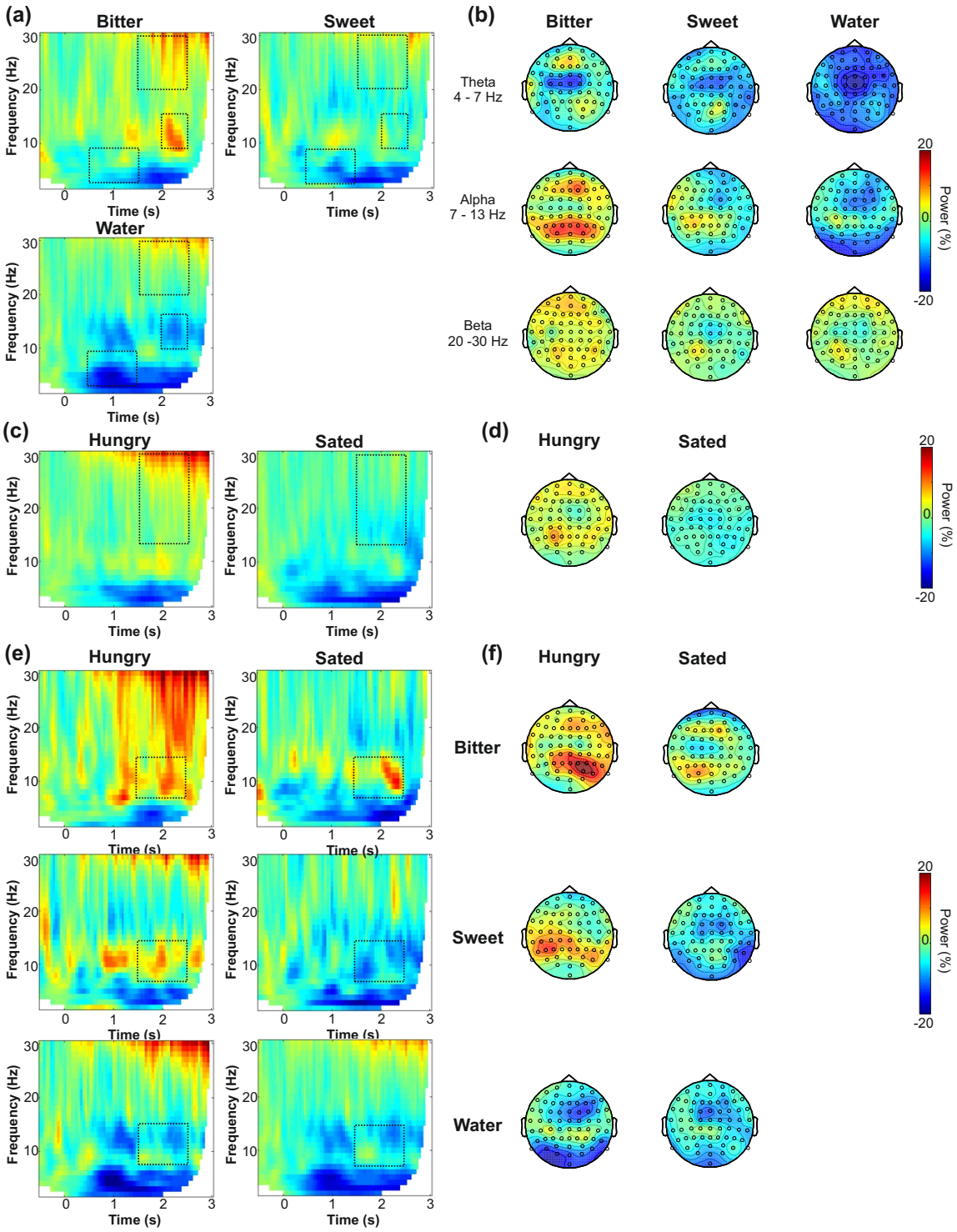
(a)
Bitter
Sweet
Water
Frequency (Hz)
Time (s)
(b)
Bitter
Sweet
Water
Theta
4 - 7 Hz
Alpha
7 - 13 Hz
Beta
20 -30 Hz
Power (%)
(c)
Hungry
Sated
(d)
Hungry
Sated
(e)
Hungry
Sated
(f)
Hungry
Sated
Bitter
Sweet
Water

Fig. 4.6. Time-frequency spectrographs (TFR plots) across all electrodes depicting the power (%) and frequency of oscillations with significant effects indicated by boxes, and topographic head plots for each effect observed in the ERD/S analysis. (a) TFR plots indicating the effects of taste on theta-(0.5 – 1.5 s), alpha-(2 – 2.5s) and beta-band (1.5 -2.5 s) oscillations. (b) Topographic headplots of the effects of taste on theta-, alpha- and beta-band oscillations. (c) TFR plots indicating the effects of hunger and satiation on beta-band oscillations (1.5 – 2.5 s). (d) Topographic headplots of the effects of hunger and satiation on beta-band oscillations. (e) TFR plots indicating the interaction effects of hunger and taste satiation on alpha-band oscillations (1.5 – 2.5 s). (f) Topographic headplots of the effects the interaction effects of hunger and taste satiation on alpha-band oscillations (1.5 – 2.5 s).

4.5. Discussion

In this chapter we examined the temporal, source localised and oscillatory processing of taste in relation to hunger state. EEG data across all electrodes, time points and frequency bands were evaluated subsequent to the onset of bitter, sweet and water tastants when participants were hungry and sated. Behavioural data revealed that, aside from sweet taste - which showed decreased pleasantness ratings when sated, each taste was evaluated similarly for pleasantness, intensity and arousal in both hungry and sated states. The ERP data revealed that taste was processed both early (90 – 160 ms) and late (800 – 1500 ms) epochs in left-frontal regions, and later (500 – 1500 ms) in left central-parietal processing regions. These effects were respectively localised to the left cingulate cortex and parietal cingulate cortex, where bitter and sweet tastes evoked greater current densities. Effects of hunger were observed in the same left-frontal (200 – 600 ms) and left central-parietal regions (100 – 1500 ms), also localised to the cingulate cortex. Hunger state and taste interacted in a late epoch (1000 – 1500 ms) in the left-frontal area, with reduced ERP amplitude to sweet taste in the hungry condition compared with the sated condition. The oscillatory data showed theta-, alpha- and beta-band modulations in response to the different tastants. Hunger accentuated frontal beta-band oscillations, and hunger and taste interacted on left-central alpha-band oscillations.

Taste

Contrary to our preliminary investigation (Chapter Two, section 2.3) and data from Chapter Three, bitter tastes were rated as more intense than sweet tastes, thus we cannot rule out effects of intensity on the EEG data. This may be due to the decreased stimuli set employed in this study resulting in increased discrimination between tastes.

ERP findings indicated that taste was first processed in an early epoch (90 – 160 ms) in the left frontal region, with bitter and sweet tastes evoking greater amplitude augmentations than water. The timing of this effect corresponds with our findings in Chapter Three, which indicated that more intense tastes receive greater processing resources than neutral tastes at early processing stages (see also Hummel et al., 2010b; Ohla et al., 2010). Later, in the same region (800 – 1500 ms), and also in the left central-parietal region (500 – 1500 ms), bitter and sweet tastes again show

greater amplitude compared with water. Previously, we found that these later effects can be attributed to hedonic stimulus characteristics, with hedonically positive and negative tastes showing similarly enhanced amplitudes compared with neutral tastes (Chapter Three). The present findings also relate to the wider literature which highlights later processing epochs to be dedicated to the coding of affective characteristics (e.g., Calvert, 2001; Dematte, Sanabria, & Spence, 2008; Hajcak et al., 2010b; Ohla et al., 2012), but also the difficulty in distinguishing between equally arousing hedonically positive and negative responses in ERP data (Hajcak et al., 2010b).

In this investigation, however, the source of the taste effects were localised to the cingulate cortex, rather than the PGC as observed in Chapter Three. The cingulate cortex is largely associated with emotional processing (see Bush, Luu & Posner, 2003, for a review), which likely explains the increased current densities observed for the more emotive bitter and sweet stimuli. Moreover, this limbic region has been associated with taste processing under hungry conditions (e.g., Hasse et al., 2009) suggesting that the emotive value of tastants is critical during this state.

Within the ERD/S data, taste modulated theta (4 – 7 Hz), alpha (7 – 13 Hz) and beta-band (13 – 30 Hz) oscillations. In relation to theta-band activity (0.5 – 1 s), all tastes evoked centralised theta-ERD, consistent with the findings from Chapter Three. However, in the current data, greater frontal theta ERS in response to bitter tastes also emerged. Theta-ERS has been associated with an increase in sub-cortical activity in regions associated with memory and emotion (e.g., Lou et al., 2013). In particular, consistent findings suggest that during emotional arousal, neurons in the amygdala produce theta activity (see Pare, 2003), and right sided theta-ERS occurs for unpleasant stimulation (e.g., Aftanas & Golocheikine, 2001), which may explain our finding of greater theta-ERS in response to bitter stimuli.

Taste also modulated right-frontal alpha-band activity and bilateral-frontal beta-band activity (2 – 2.5 s). Water and sweet tastes evoked alpha-band ERD (as observed in Chapter Three), contrasting with bitter responses which showed alpha ERS. Bitter tastes also evoked beta-ERS compared with water and sweet taste, which evoked little change. Curiously, beta-band ERD is generally associated with increased attention and arousal (Başar, Başar-Eroglu, & Karakaş, 2001; Keil et al., 2001; Klimesch, 1999; Klimesch, Doppelmayr, & Russegger, 1998), whereas beta-band ERS is associated with the suppression of cortical activity (Klimesch et al.,

1998). Given this relationship, we could expect that the more arousing tastes, such as bitter and sweet, would evoke greater ERD, which we have previously observed in beta-band responses (Chapter Three).

However, there are indications that beta-ERS is linked with active cortical inhibition (Klimesch et al., 1998). Moreover, it has been shown that right-frontal beta-ERS is associated with avoidance related measures (Coan & Allen, 2004; Harmon-Jones et al., 2010; Schutter et al., 2007). Thus, it may be that bitter taste evokes avoidance mechanisms. Given that beta-ERS responses to bitter tastes were absent in the sated condition [Figure 4.4 (f)], it is possible that being in a fasted state results in the cortical inhibition of responses to tastants, particularly aversive tastes, as has been observed in those actively suppressing the motivation to eat (Yoshikawa et al., 2014). Given the limited literature on neural oscillations in appetite and taste processing, this interpretation is purely speculative and requires much more research.

Hunger state

Behaviourally, hunger had little impact on taste intensity, pleasantness or arousal ratings, contrary to previous reports (e.g., Moskowtz et al., 1976; Zverev, 2004). Hunger did, however, greatly affect the EEG findings. Hunger enhanced ERP amplitude in left-frontal and parietal regions. Frontally, hunger evoked greater negative amplitude between 200 – 600 ms, but matched the amplitude of the sated condition between 1000 – 1500 ms. These effects were source-localised to the cingulate cortex but no differences between hunger conditions were observed. Parietally, the amplitude enhancement evoked by hunger was maintained across the 1500 ms epoch, although the greatest current densities at this time were observed in the right parietal region where no differences between hunger conditions were observed.

Previous studies have reported increased responses in the hypothalamus, amygdala and hippocampus to tastes when hungry (Hasse et al., 2009). It is therefore possible that the current results reflect differences in these regions. Moreover, the left hemispheric specificity for hungry compared with sated states was reported in a recent EEG study, with left posterior sites showing greater amplitude deflections for hungry participants when viewing food pictures (Stockburger et al., 2008), suggesting that hunger augments EEG amplitude in the presence of food related

stimuli. The current study goes someway to support these results as hunger provoked enhanced left-lateralised EEG amplitudes in response to tastants.

Hunger was also found to have a substantial effect on beta-band oscillations in both bilateral-frontal and bilateral-parietal regions (1.5 – 2.5 s). Considerably greater beta-ERS was observed in the hungry condition, compared with little beta-band activity in the satiated condition. As discussed earlier, this may be related to cortical inhibition as a result of an avoidance mechanism (Coan & Allen, 2004; Harmon-Jones et al., 2010; Schutter et al., 2007) and a tentative explanation may be that hungry participants were actively suppressing the motivation to eat in the fasted condition (Yoshikawa et al., 2014).

Taste × hunger state

Behaviourally, sweet taste was rated as less pleasant when sated. This finding is in line with the alliesthesia effect (Cabanac, 1971) in that the sweet solution became less palatable after a sweet meal. This observation differs from those obtained by Moskowitz et al. (1976), who reported that sated individuals are unable to discriminate affect and intensity. Rather, our data suggest that this discrimination process may be increased following satiation and this may be specific to palatable tastes. No differences were reported in pleasantness for bitter and water tastes, suggesting that hunger and satiety do not affect taste ratings across the board, only taste that are nutrient rich. However, the current investigation only examined taste ratings following a sweet meal (or no meal), it would be interesting to see if the same effects were observed following a savoury meal, or whether pleasantness ratings for sweet tastes would increase in line with sensory-specific satiety (e.g., Rolls, 1981).

Interactions between hunger and taste occurred in late (1000 – 1500 ms) left frontal ERP data, and left central-parietal alpha oscillations. In both cases, the interaction was largely a result of differences in the processing of sweet taste between the hungry and sated conditions. ERP responses to sweet taste were greater when participants were full, while bitter showed equal amplitude in both conditions. Water evoked greater responses when hungry, but this may be due to the increased ratings of thirst that were recorded in the hungry condition. The greatest current density at this time was observed in the right middle frontal gyrus, where responses to taste when full were attenuated compared with those obtained when hungry. This

fits with the effects observed by Hasse et al. (2009) and Eldeghaidy et al., (2016) where attenuated activations were observed in the nearby OFC and reward areas when participants were sated. However, there were no hunger state and taste interactions in this region.

In the oscillatory data, left-lateralised alpha-band ERD occurred for sweet in the sated condition, whereas ERS occurred in the hungry condition, suggesting that alpha responses to sweet tastes are inhibited when hungry and enhanced when full. In Chapter Three, we observed left-lateralised alpha-band ERD in response to the discrimination of taste hedonicity, which was greatest for pleasant, sweet tastes. The same effect occurs here in the sated condition, but appears to be suppressed in the hungry condition. As previously mentioned, it may be the case that the specific neural mechanisms involved in the evaluation of taste hedonicity may be attenuated when hungry. Sweet tastes are almost always pleasant, but when hungry, attention to the nutritional value of a food may take precedence over evaluating its palatability, perhaps as a part of a process of promoting survival when food resources are limited (e.g., Benson, 1977; Sheppard, 1975; Sherratt, Speed & Ruxton, 2003; Strygley & Kingsolver, 1998). Alternatively, our observations may reflect the operation of 'wanting', in which the incentive motivational value of the taste matches a specific motivational state (hunger) and is distinct from its hedonic impact ('liking') (e.g., Berridge, 2004). The fact that changes to the ERP and alpha-band representation of bitter tastes under hungry conditions did not occur, suggests that this discriminative process may be specific to nutritionally relevant tastes, and not a general mechanism for all tastes.

Limitations

We recognise that, as in Chapter Three, we again failed to produce distinctive peak ERP components: rather, clusters of differential activity were observed. This outcome was partly anticipated, although attempts were made to avoid this. We speculate that the stimuli repetitions required to generate ERP peaks were not met in this investigation. Moreover, despite the reduced testing duration compared with that of Chapter Three, we still experienced excessive head movements, which resulted in the loss of many trials. We suspect that increasing stimuli repetitions and reducing testing durations further will eliminate this problem; this is corroborated by our findings in the next chapter (Chapter Five). Nevertheless, an ERP analysis was

performed and even in the absence of typical peak components, we were able to establish specific patterns of amplitude activation in the processing of taste and hunger information.

Additionally, there is a possible confounding influence of using a sweet meal in relation to the measure of responses to sweet taste in the two conditions. This may be problematic in relation to any interpretation of the data, specifically distinguishing between caloric-satiety and possible sensory-specific satiety phenomena.

Conclusions

Both gustation and appetite involve complex neurological and physiological mechanisms that interact in the initiation, maintenance and termination of eating behaviour. The current investigation is one of the first to examine the relationship between hunger and gustation using EEG, and the data go some way to characterise the temporal and oscillatory dynamics of this interaction in normal- slightly over-weight individuals.

Taste processing was characterised by early and late left-ERP components showing increased responses for more intense and arousing tastants, and respective specific left-lateralised alpha-band ERD or centralised theta-band ERD in response to sweet and bitter tastes. Contrary to Chapter Three, the cingulate cortex was most activated during this processing, suggesting that hunger and satiety evoke greater activations in areas associated with reward and emotion during taste processing, compared with greater activations in PGC areas when no fasting or fed manipulations are employed. Thus, hunger state can greatly affect the processing of taste information and this should be considered in future studies.

Hunger enhanced overall ERP responses, suggestive of increased attentional resources assigned to taste stimuli during states of hunger (Stockburger et al., 2008). The oscillatory data, however, suggests that hunger results in frontal cortical inhibition, particularly for aversive tastes and this may be associated with an avoidance mechanism, although this needs further investigation.

Sweet tastes were found to be less pleasant when sated and neural signals in response to sweet taste were enhanced after satiating on a sweet meal. Our findings in Chapter Three, in relation to the processing of sweet tastes, mirror those observed

here in the sated condition, thus it can be speculated that hunger attenuates neural mechanisms involved in the evaluation of taste hedonicity and that this may be specific to nutritionally relevant taste (e.g., Moskowitz et al., 1979).

Contrary to the Jacquin-Piques et al. (2016) EEG investigation, we found that hunger and satiety greatly influence the processing of tastes, to the extent that greater activations in regions associated with motivation and reward, rather than PGC regions were observed. In particular, the state of hunger was found to enhance ERP amplitudes as well as increase oscillatory synchronisations associated with cortical inhibition. Thus, without employing stringent fasting and fed states, these differences may be obscured. Although, the present results do suggest that when measuring taste responses, careful consideration of hunger state should be employed.

Chapter Five: The Influence of Expectancy on Taste Processing

5.1. Abstract

Taste expectations have been shown to affect sensory and hedonic ratings of tastants, but it is unclear whether they have the ability to shape sensory experiences at a perceptual level. In this chapter we explored the neural underpinnings of the taste-expectancy relationship. Using a trial-by-trial cueing paradigm, combined with ratings of intensity and pleasantness, participants were validly or invalidly cued to anticipate either a low- or high-concentration of a sweet taste. EEG was recorded relative to the onset of the tastant and the ERPs, ERP source-localisation and ERD/S were examined. Intensity ratings for high-sweet tastes decreased when the taste was invalidly cued (participants were cued to expect a low-sweet taste) and increased for low-sweet tastes when they were invalidly cued (participants were cued to expect a high-sweet taste). Articulated gERPs were observed and primary gustatory cortices were activated. P1 amplitudes in the left parietal region, and alpha-band ERD in the right parietal region shifted toward responses that would be engendered were the taste stimulus to match expectation. The data demonstrate that prior expectations not only modify subjective intensity ratings of sweet taste, but can also modulate early sensory representations.

5.2. Introduction

The goal of perception is to take information from the sensory world and make sense of it. To do this, we must combine information from our sensory environment with our own experiences, beliefs and expectations. (Gibson, 1966, 1979; O'Regan & Noe, 2001; Rosch, Varela & Thompson, 1991). Taste perception, in particular, is influenced by a variety of factors beyond primary sensory information. We demonstrated in Chapter Four the impact that the physiological states of hunger and satiety can have on the processing of tastes. In this chapter we explore the effects of cognitive factors on taste processing. A number of behavioural studies have shown that characteristics such as colour, texture, odour, price and fat content have been shown to give rise to expectations that influence subsequent flavour evaluations (e.g., Cardello & MacFie, 2007; Koch & Koch 2003; Lee, Frederick, & Ariely, 2006; Levitan et al., 2008; Shankar et al., 2009; Yeomans et al., 2008; Zampini et al., 2008; Zellner & Durlach, 2003). This chapter explores whether such expectations can directly affect primary taste processing, thus indicating a modulation of responses at a perceptual level.

Expectations refer to the possession of prior information about the possible or probable forthcoming events in the sensory environment (Summerfield & Egner, 2009). This information is known to modulate brain processes. Generally, expected stimuli will evoke decreased brain activations compared with stimuli that are unexpected (see Segaert et al., 2012, for a review) and elicit decreased N400 ERP components, compared with their unexpected counterparts (see Kutas & Federmeier, 2011, for a review). These responses are argued to have developed in order to reduce cognitive load, providing an organism with an efficient information processing system (Kutas & Federmeier, 2011).

While it is well known that expectations can influence judgements of the quality, intensity and the pleasantness of a tastant (Cardello & MacFie, 2007; Koch & Koch 2003; Lee, Frederick, & Ariely, 2006; Levitan et al., 2008; Shankar et al., 2009; Yeomans et al., 2008; Zampini et al., 2008; Zellner & Durlach, 2003), the question of how expectations can influence the subjective perception of tastes is less well understood. The Assimilation-Contrast Model of expectation (Heider, 1944; Sherif & Hovland, 1961) holds that subjective ratings can follow, or assimilate to the level of prior expectation, providing that the contrast between expectation and

experience is minimal (low-disconfirmation, see Chapter One, section 1.8.2). Many argue that such effects take place at a perceptual level, whereby neural responses to a taste adapt to assimilate to the prior expectations (Braun-LaTour & LaTour, 2005; Hoch & Ha, 1986; Lee, Frederick & Ariely, 2006; Okamoto & Dan, 2013).

Only a limited number of studies have addressed this issue, but there is now evidence that cortical responses to taste can be modulated by expectation. As discussed in Chapter One (section 1.8.3), Nitschke et al. (2006) and Sarinopoulolos et al. (2006) found that when participants were presented with a cue that led them to believe that an upcoming bitter quinine taste would be less distasteful than it actually was, they reported it to be less aversive than when they received accurate information. Moreover, fMRI data showed that bitter taste activated the bilateral PGC less strongly when preceded by a mildly aversive cue, than by a highly aversive cue. However, no reliable changes in cortical activation as a function of pleasant taste or cue-condition were detected, suggesting that the effects reported with quinine solutions reflect specific processing of aversive stimuli and a lack of generalization of the expectancy effect to other tastes. However, in this study, subjective ratings were obtained for pleasantness but not intensity, thus it may be the case that the expectancy effects were related to the perception of intensity and not directly to changes in pleasantness (Okamoto & Dan, 2013).

A later study by Woods et al. (2011) has provided evidence of assimilation effects in relation to sweetness. In that study, a 'very sweet' textual cue both enhanced subjective ratings of intensity of a diluted orange juice drink and increased activations in the PGC. Interestingly, a complementary devaluation of ratings was not observed when an undiluted juice was preceded by a 'less sweet' cue, and there was no alteration of cortical activation.

The neural underpinnings of the taste-expectancy relationship have largely been examined using fMRI (e.g., Grabenhorst et al., 2007; Nitschke et al., 2006; Plassmann et al., 2008; Sarinopoulos et al., 2006; Woods et al., 2011). While fMRI has high spatial resolution, allowing for the localisation of neural regions in which certain processes take place, EEG has high temporal resolution. ERPs allow for the investigation of sequential taste processing and can provide important information about whether effects take place at early perceptual stages of processing (P1, N1) or later cognitive stages (> 300 ms; LPP), with source-localisation methods estimating

the regional origin of these effects. In addition, examining ERD/S in oscillatory activity would provide a novel insight into how expectancies affect the perceptual and cognitive processing of tastes.

In the following experiment, we recorded EEG activity to examine cue-induced expectancy effects on the processing of sweet sucrose solutions. We employed a trial-by-trial cueing paradigm, combined with ratings of pleasantness and intensity for both anticipated and actual taste stimuli. On-screen cues were presented to indicate, either validly or invalidly, that there was a high probability that a subsequent taste stimulus would have either a low- or high-sweet taste. Based on results from Chapters Three and Four, we again reduced the stimuli set, this time to just two tastants and increased the repetitions to 50 per tastant, resulting in a reduced testing time and decreased likelihood of noise. Thus we expected to observe fully articulated ERP peaks. In order to reduce testing time further, we eliminated the arousal measure. In Chapter Four arousal ratings increased commensurately with intensity so distinguishing between effects of intensity and arousal was not possible, therefore, this measure was deemed unimportant to the current investigation. We also controlled for effects of hunger state (Chapter Four) by requesting participants to eat their normal breakfast or lunch prior to testing.

We predicted that evidence of assimilation to the expected taste would be found in subjective ratings of taste stimuli, and that such effects would have correlates in the temporal analysis of EEG data. More specifically, if expectation influences the perceptual processing of taste, we expect taste-expectancy interactions on early sensory evoked ERP components originating from PGC regions, and on the alpha and beta oscillatory components that were associated with sweetness and intensity processing in Chapters Three and Four. If expectation influences evaluative processing instead of, or in addition to sensory processing, then taste-expectation interactions would be predicted to occur in later (>300 ms) processing stages in secondary gustatory regions.

5.3. Materials and Methods

5.3.1. Participants

Sixteen female participants, aged 19 -31 years (M = 24.75, SD = 3.9) took part in the study. Participants were normal- to over-weight (BMI range 19.5 – 27.9; M = 23.0, SD = 2.45)[2]. All participants were pre-screened to ensure they were non-smokers, non-diabetic, had no food allergies or intolerances or taste disorders, and were not taking medications or suffering illnesses that could interfere with their gustatory or olfactory perception. All participants gave informed consent and the study was conducted in accordance with the Code of Ethics of the World Medical Association (Declaration of Helsinki), and was approved by the University of Liverpool Ethics Committee.

5.3.2. Design

A 2 × 2 within-subjects design was employed. The independent variables were taste (low-sweet, high-sweet) and expectation (valid cue, invalid cue). The dependent variables were ERP amplitude, current densities at ERP latencies and ERD/S power.

5.3.3. Taste stimuli

The selection of taste stimuli is described in Chapter Two (section 2.3). We selected a low concentration of sucrose (0.05 M, low-sweet) and a high concentration of sucrose (0.3 M, high-sweet) for their discernibly different taste intensity ratings (p < .001), but not too different pleasantness ratings or taste quality so as to induce any expectancy-contrast effects (e.g., Wilson & Klaaren, 1992: see also Chapter One, section 1.8.2).

5.3.4. Measurements

Appetite was measured using a six part VAS scale (0 – 100) measuring hunger, fullness, desire to eat, satisfaction, nausea and thirst (Flint et al., 2000; Rolls et al., 1999). Appetite scores did not interact with EEG findings (ps > .08). Before and during testing, each taste was rated for pleasantness and intensity using the LAM (Schutz & Cardello, 2001) and the gLMS (Bartoshuk et al., 2004), respectively. As shown in Figure 5.1 (a), initial ratings indicated that the high-sweet taste was evaluated as more pleasant (M = 17.37, SD = 22.7) than the low-sweet taste (M = 5.31, SD = 12.31), t (15) = 2.09, p = .05, and more intense (M = 46.19, SD = 20.13) than the low sweet taste (M = 10.75, SE = 6.39), t (15) =6.41, p < .001. However, as

[2] Variations in BMI had no effect on behavioural of EEG data (ps > .29)

can be seen from Figure 5.1 (b), the mean pleasantness ratings for the high-sweet taste taken during the experiment (M = -10.37, SE = 7.66) were significantly lower than those taken prior to the study, t (15) = 2.85, p = .012, showing a steady decline over trials [Figure 5.1 (c)]. Moreover, high-sweet and low-sweet tastes were rated as equally mildly unpleasant during the experiment (p = .392). Intensity ratings showed no change (p = .257).

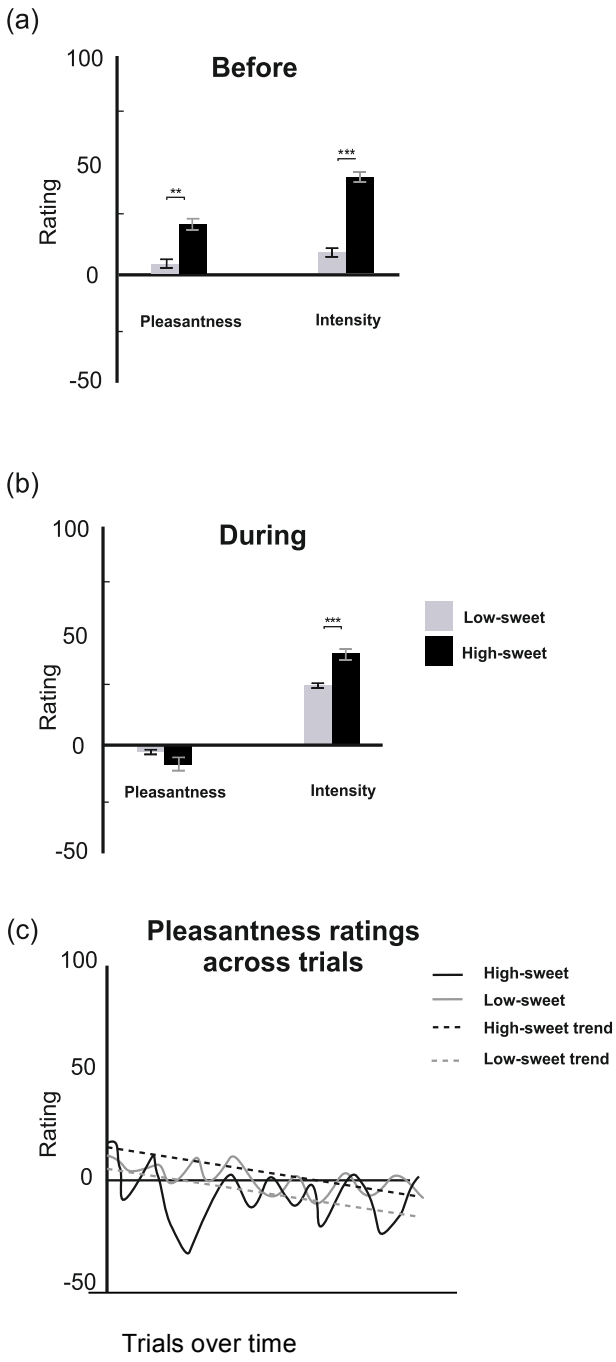

Fig. 5.1. Bar charts representing the mean pleasantness and intensity ratings (a) taken before the study and (b) taken during the course of the experiment. Error bars indicate standard error. (c) A line graph indicating the pleasantness ratings across the trials**.** Asterisks indicate significant differences: ** p< .01; *** p<001.

5.3.5. Stimulus presentation

The taste stimuli were presented using the same gustometer method as described in Chapter Three. Prior to the administration of the taste, however, the participants were presented with either a blue or yellow fixation cross on a computer monitor, indicating that there was a 70% probability that the next taste sample would be either low-sweet (yellow cue) or high-sweet (blue cue). Each participant then rated the *expected* pleasantness and intensity of that taste using onscreen LAM (Schutz & Cardello, 2001) and gLMS (Bartoshuk et al., 2004) scales. Participants were instructed to wait as the experimenter monitored a video link and EMG data, initiating taste delivery when the participant was making no movements or swallowing motions. Participants were required to hold the taste in their mouth for 3 seconds whilst remaining still, before rating the tastes for *actual* intensity and pleasantness. Each tasting was followed by a 4-second (2 ml) distilled water rinse. The ratings and wait period allowed for an inter-stimulus interval of ~ 30 seconds, so controlling for habituation and adaptation (Evans et al., 1993). The order of the taste samples was randomised. Overall, both of the stimuli were repeated 50 times (100 trials, separated into 4 blocks of 25 trials). Participants were instructed that the cues would validly predict the taste 70% of the time, and thus were invalid for 30% of the trials. Figure 5.2 summarises the trial procedure.

5.3.6. Procedure

All participants began the testing procedure between 09:00 and 10:00, or between 12.30 and 13:00, and were required to eat their normal breakfast or lunch prior to testing, so as to ensure there were no confounding effects of hunger on the EEG data (see Chapter Four). Participants completed the appetite questionnaires and tasted and rated a 10 ml sample of each test solution for their intensity and pleasantness using the LAM and gLMS. The EEG equipment was fitted to the participant who was then seated in the experimental chamber. Participants completed 4 practise trials, comprising 2 high-sweet validly cued trials and 2 low-sweet validly cued trials. The practice also served as a prime for associating the tastes with the coloured fixation crosses. Participants then completed the experimental trials, with stimuli delivered and LAM and gLMS ratings as described above. Overall, the experiment took ~ 1 hour to complete.

5.3.7. Electrophysiological Measures: ERP, sLORETA and ERD/S

The ERP, sLORETA and ERD/S data were recorded and cleaned using the same methods described in Chapter Three. An average of 9.2 (*SD* = 3.4) noise components were removed using ICA. No participant incurred a loss of > 50% of trials in any one condition, thus no participant data were removed from EEG analyses. There was an average (mean ± *SD*) of 36.13 (± 5.6) high-sweet taste trials remaining and 37.69 (± 3.70) low-sweet trials retained for each participant following pre-processing procedures. There were no significant differences in the trials retained for each condition ($p = .849$).

5.3.8. Statistical Analysis

As with Chapters Three and Four, we performed three types of analysis: the standard time-domain averaging technique to examine ERPs, sLORETA to examine the origin of the ERP effect and a wavelet-based TFR to analyse underlying neural oscillations in the form of ERD/S. The statistical analysis procedures for each of the EEG analyses were the same as described in Chapter Three. Once identified, the ERP clusters, sLORETAs and ERD/S clusters were evaluated for each participant, and subjected to a series of within-subjects ANOVAs with the factors: taste (low-sweet, high-sweet), expectancy (valid cue, invalid cue) and taste x expectancy.

Post hoc analyses using pairwise comparisons and Bonferroni corrections were conducted for each EEG analysis when significant effects occurred. Greenhouse-Geisser corrections were applied when statistical assumptions were not met. Where multiple significant effects occurred, results were collated to show the smallest mean difference, greatest standard error and greatest *p* values respectively (*MD*s >, *SE*s <, *p*s <). Effect sizes (ES) represent the partial η^2 value.

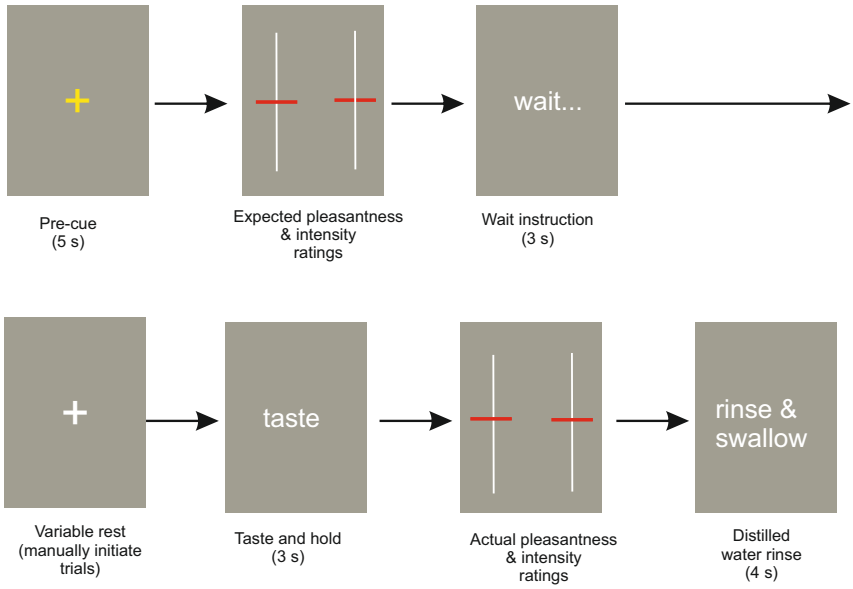

Fig. 5.2. A schematic diagram of the experimental procedure.

5.4. Results

5.4.2. Behavioural Analysis

Intensity ratings

We first examined the influence of taste and expectancy on intensity ratings taken after tasting [Figure 5.3 (a, b)] using a 2 x 2 repeated measures ANOVA. We then compared *predicted* and *actual* taste intensity ratings in validly and invalidly cued trials for each taste [Figure 5.3 (c)] using 2 x 2 x 2 repeated measures ANOVA.

There was a significant effect of taste (low-sweet, high-sweet) on intensity ratings after tasting, with high-sweet rated as more intense than low-sweet taste, F (1, 15) = 94.58, $p < .001$, ES = .86 [Figure 5.3 (a)]. Importantly, a significant interaction between taste and expectancy on intensity ratings after tasting was apparent, F (1, 15) = 41,72 $p < .001$, ES = .74 [Figure 5.3 (b)]. Invalidly cued low-sweet tastes (on trials when where high-sweet tastes were cued) were rated as more intense than validly cued low-sweet tastes. In contrast, invalidly cued high-sweet tastes (on trials when low-sweet tastes were cued) were rated as less intense than validly cued high-sweet taste

When including predicted versus actual ratings as a variable, we also observed particularly marked contrasts between predicted and actual intensity ratings for each taste when there was a mismatch between expectation and actual sweetness level, F (1, 15) = 52.68, $p < .001$, ES = .79 [Figure 5.3 (c)]. When expectations were met, ratings of predicted intensity were much more similar to ratings of the actual taste. Thus, the results indicate a successful manipulation of expectancy.

Pleasantness Ratings

As with the intensity ratings, we first examined the affect of taste and expectancy on pleasantness ratings taken after tasting using a 2 × 2 repeated measures ANOVA. We then compared *predicted* and *actual* taste intensity ratings in validly and invalidly cued trials for each taste using 2 × 2 × 2 repeated measures ANOVA.

Analysis of pleasantness ratings taken after tasting revealed no significant effects of taste (p = .329), expectancy condition (p = .321), and no interactions (p = .41).

There was also no difference between predicted versus actual ratings of taste pleasantness when there was a mismatch between expectation and actual sweetness level (p = .489). This corresponds with the previous analysis showing no differences in mean pleasantness ratings between low- and high-sweet tastes during the experiment.

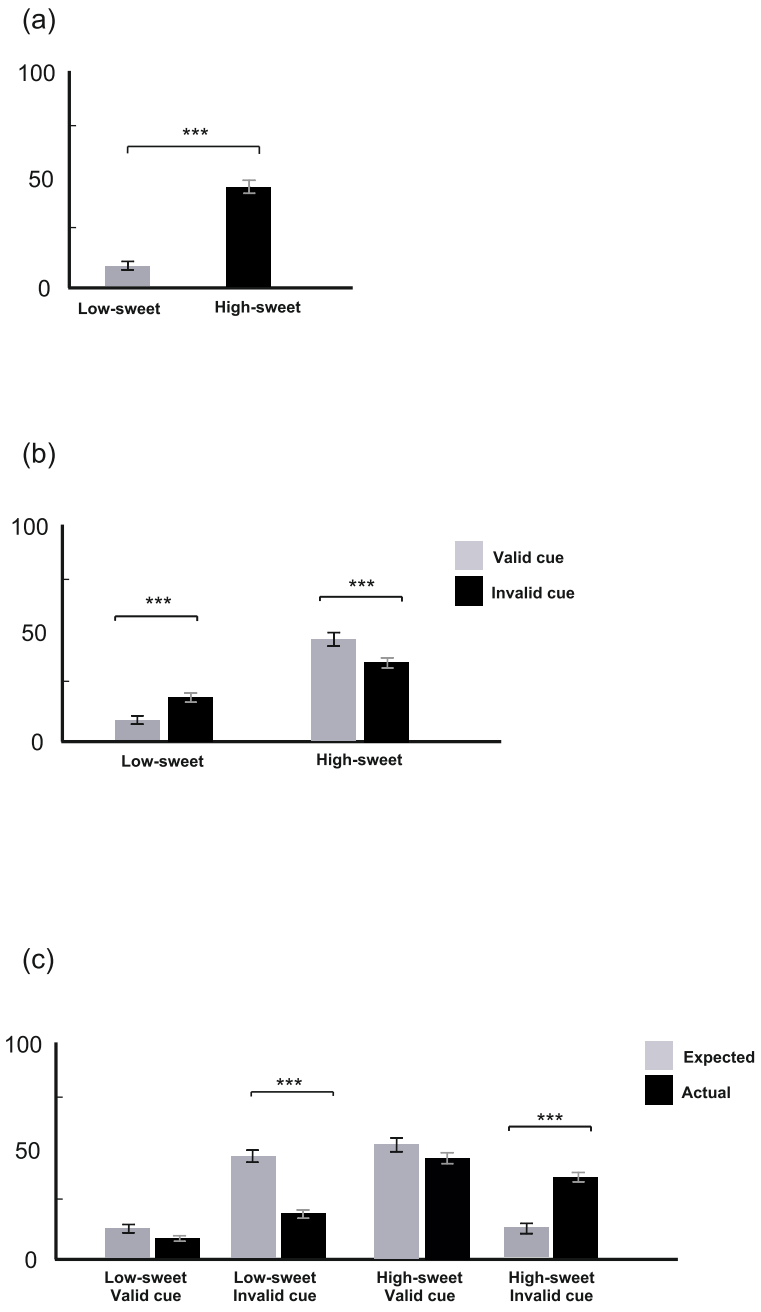

Fig. 5.3. Bar charts representing the (a) mean intensity ratings for low-sweet and high-sweet tastes taken after tasting, (b) mean intensity ratings for low-sweet and high-sweet tastes under valid and invalidly cued conditions taken after tasting, (c) mean expected and actual intensity ratings for low-sweet and high-sweet tastes under valid and invalidly cued conditions taken before (expected) and after (actual) tasting. Error bars indicate standard error. Asterisks indicate significant differences: * $p <$ .05; ** $p <$ .01; *** $p <$ .001.

5.4.3. ERP analysis

As Figure 5.4 illustrates, a fully articulated ERP waveform was observed. The data indicated distinct differences in ERP components for taste, expectancy and taste × expectancy interactions relative to the onset of the tastant (Figure 5.5). The principal effects are summarised below for each factor.

Taste

Effects of taste on ERP data occurred in right central-parietal region (Cp2, Cp4, Cp6, P2, P6). A significant effect emerged at the P1 component (80 – 120 ms), F (1, 15) = 20.15, $p < .001$, ES = .57. As can be seen from Figure 5.5 (a, b), high-sweet tastes evoked a greater P1 peak ($M = 1.09$, $SE = 0.24$) than low sweet tastes ($M = 0.78$, $SE = 0.17$). A similar effect was also detected for the P2 component (350 – 450 ms), F (1, 15) = 4.64, $p = .048$, ES = .57, when the high-sweet taste again evoked greater peak amplitude ($M = 1.66$, $SE = 0.42$) than the low-sweet taste ($M = 1.2$, $SE = 0.36$). The ERP plot [Figure 5.5 (a)] shows this effect continuing between 650 – 1000 ms, although this did not reach significance ($p = .121$).

Expectancy

ERP effects of expectancy (valid/invalid cue) occurred in right- (Af4, Af8, F2, F4) and left- (Af3, Af7, F1, F3, F5, Fc1) frontal regions. As can be observed from Figure 5.5 (c, d), the valid and invalidly cued conditions evoked equal amplitudes at both left- ($p = .66$) and right-frontal ($p = .95$) P1 (150 – 250 ms) components. However, at the N400 component (350 – 450 ms), a significant effect emerged in the right frontal region, where tastes that were validly cued ($M = -1.78$, $SE = 0.37$) elicited a reduced peak compared with those that were invalidly cued ($M = -2.39$, $SE = 0.28$), F (1, 15) = 4.93, $p = .042$, ES = .25 [Figure 5.5 (c, e)]. From Figure 5.5 (d, e), we can see that an opposite effect occurred in the left-frontal region at this time, with the validly cued tastes eliciting a greater N400 peak ($M = -1.48$, $SE = 0.30$) than the invalidly cued tastes condition ($M = -0.77$, $SE = 0.31$), F (1, 15) = 4.47, $p = .05$, ES = .23. In both the left and right frontal regions, these differences disappeared for the remainder of the epoch ($ps > .188$).

Taste × expectancy interactions

Taste × expectancy interactions on ERP data were observed in the left parietal region [P9, Po3, Po7; F (1, 15) = 5.29, p = .036, ES = .26]. As illustrated in Figure 5.5 (f, g), at the P1 component (100 – 150 ms) when low-sweet tastes were validly cued, they evoked a greater P1 amplitude (M = 1.08, SE = 0.35) than when they were invalidly cued (high-sweet taste cued), (M = 0.14, SE = 0.35). In contrast, invalidly cued high-sweet tastes (low-sweet taste cued) evoked an increase in amplitude (M = 2.28, SE = 0.45) compared with validly cued high-sweet (M = 0.95, SE = 0.45).

Taste × expectancy interactions were also observed in the left-frontal region (FT7, Fc5, Fc1) in relation to the LPP between 500 – 1500 ms, F (1, 15) = 6.71, p = .02, ES = .31 [Figure 5.5 (h, i)]. When low-sweet tastes were invalidly cued they evoked greater amplitude (M = 1.05, SE = 0.50) than when they were validly (M = -0.17, SE = 0.50). High-sweet tastes, on the other hand, evoked similar amplitude when validly cued (M = -0.16, SE = 0.49) or invalidly cued (M = -0.46, SE = 0.42).

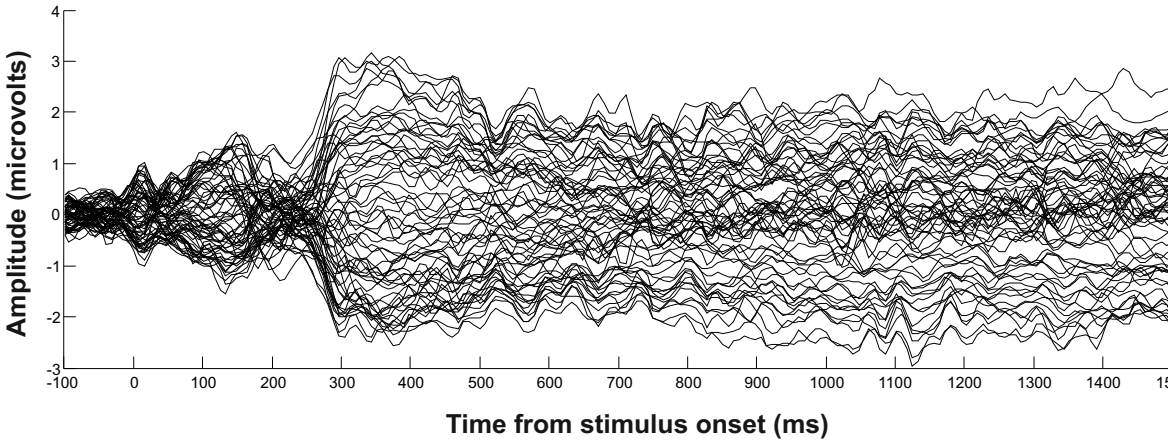

Fig. 5.4. Butterfly plot demonstrating the average ERP waveform across conditions for each electrode (separate lines) from the onset of the tastant.

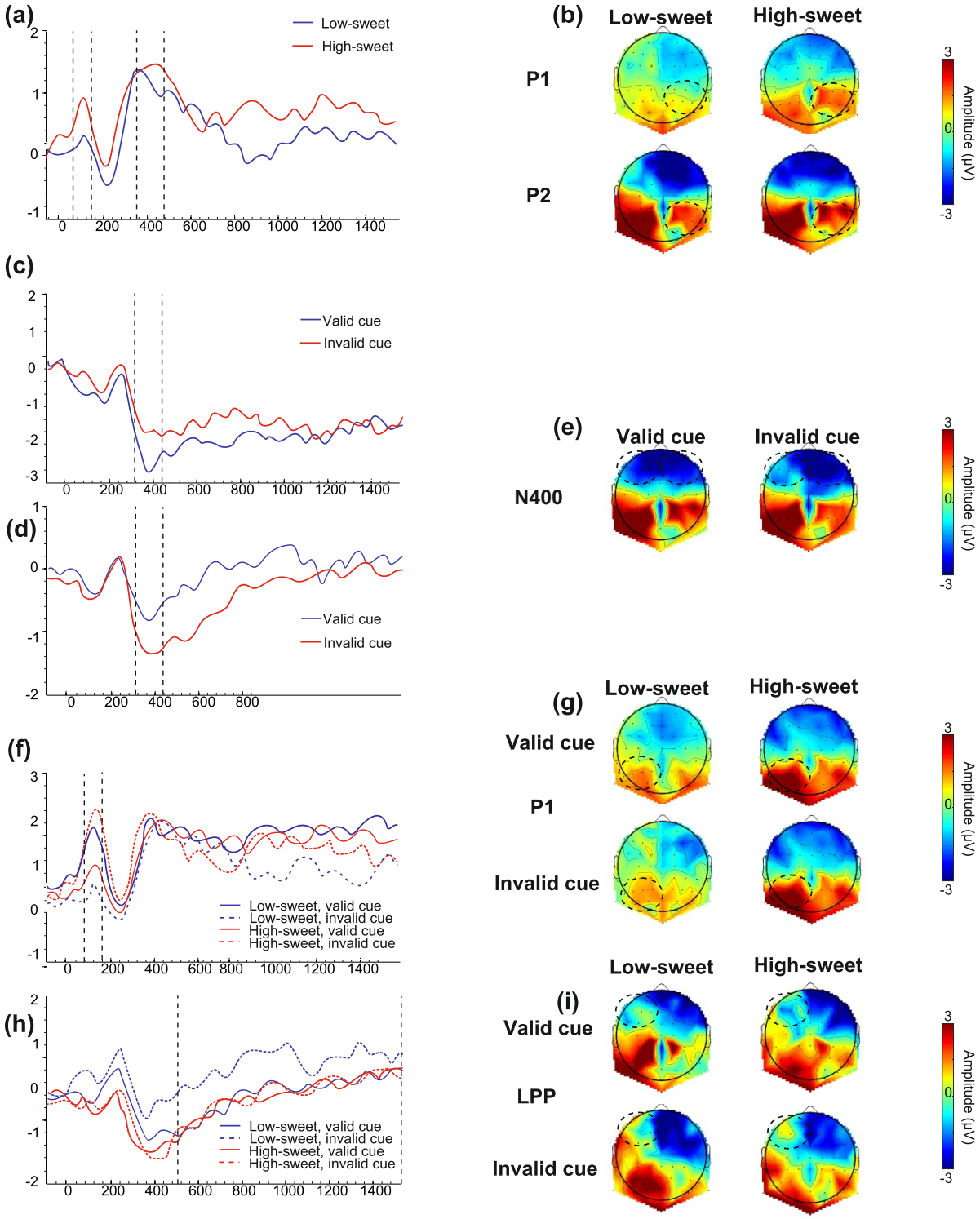
(a)
Low-sweet
High-sweet
(b)
Low-sweet
High-sweet
P1
P2
Amplitude (μV)
(c)
Valid cue
Invalid cue
(d)
Valid cue
Invalid cue
(e)
Valid cue
Invalid cue
N400
(f)
Low-sweet, valid cue
Low-sweet, invalid cue
High-sweet, valid cue
High-sweet, invalid cue
(g)
Low-sweet
High-sweet
Valid cue
P1
Invalid cue
(h)
Low-sweet, valid cue
Low-sweet, invalid cue
High-sweet, valid cue
High-sweet, invalid cue
(i)
Low-sweet
High-sweet
Valid cue
LPP
Invalid cue

Fig. 5.5. Temporal ERP amplitude plots with vertical dashed lines indicating intervals with significant main effects of condition, and topographic head plots with colour bars representing amplitude (μV). (a) Significant ERP effects of taste in the left frontal region (Cp2, Cp4, Cp6, P2, P6) at P1 (80 – 120 ms) and P2 (350 – 450 ms). (b) Topographic headplots indicating taste effects between at P1 and P2. (c) N400 ERP effects of expectancy (valid/invalid cue) in right- (Af4, Af8, F2, F4) and (d) left- (Af3, Af7, F1, F3, F5, Fc1) frontal region. (e) Topographic headplots indicating expectancy effects at N400. (f) Significant ERP amplitude effects of taste × expectancy in the left parietal region (P9, Po3, Po7) at P1. (g) Topographic headplots indicating taste × expectancy effects at P1. (h) Significant ERP effects of taste × expectancy in the left-frontal region (FT7, Fc5, Fc1) in the LPP (500 – 1500 ms). (i) Topographic headplots indicating taste × expectancy effects at the LPP.

5.4.4. sLORETA analysis

Figure 5.6 illustrates the sLORETA source estimates of each of the ERP latencies and the current densities for each condition. The principal sources are summarised below for each factor.

Taste

During the P1 taste latency (80 – 120 ms) activations were observed in the right-insula cortex [TAL, $x = 33$, $y = 4$, $z = 21$; Figure 5.6 (a)]. There was a significant effect of taste on current density, F (1, 15) = 5.57, $p = .023$, ES= .27, which was significantly increased in responses to high-sweet ($M = 1.89$, $SE = 0.35$) compared with the low-sweet tastes [$M = 1.22$, $SE = 0.26$; Figure 5.6 (b)]. At the P2 latency (350 – 350 ms) the greatest activations were again estimated to occur from the right insula cortex [TAL $x = 33$, $y = 4$, $z = 21$; Figure 5.6 (c)] but no differences in current densities for the taste conditions emerged [$p = .633$; Figure 5.5 (d)].

Expectancy

During the N400 expectancy latency (350 – 450 ms), activations were estimated to be greatest in the right insula cortex [TAL, $x = 33$, $y = 4$, $z = 21$; Figure 5.6 (e)]. There was a significant effect of expectancy on current density, F (1, 15) = 8.86, $p = .009$, ES = .37, where invalidly cued tastes elicited a greater current density ($M = 1.55$, $SE = .46$) than validly cued tastes [$M = 0.75$, $SE = .21$; Figure 5.6 (f)].

Taste × expectancy interactions

During the P1 taste × expectancy latency (100 – 150 ms) a cluster of activation was observed in the right insula cortex [TAL, $x = 33$, $y = 4$, $z = 21$; Figure 5.6 (g)], but the taste x expectancy interaction on current densities failed to reach significance [$p = .08$, Figure 5.6 (h)]. During the LPP latency (500 – 1500 ms), a cluster of activation was observed in the right anterior cingulated cortex [ACC; TAL, $x = 8$, $y = -11$, $z = 30$; Figure 5.6 (i)]. However, there were no taste × expectancy interactions on current densities [$p = .915$; Figure 5.5 (k)].

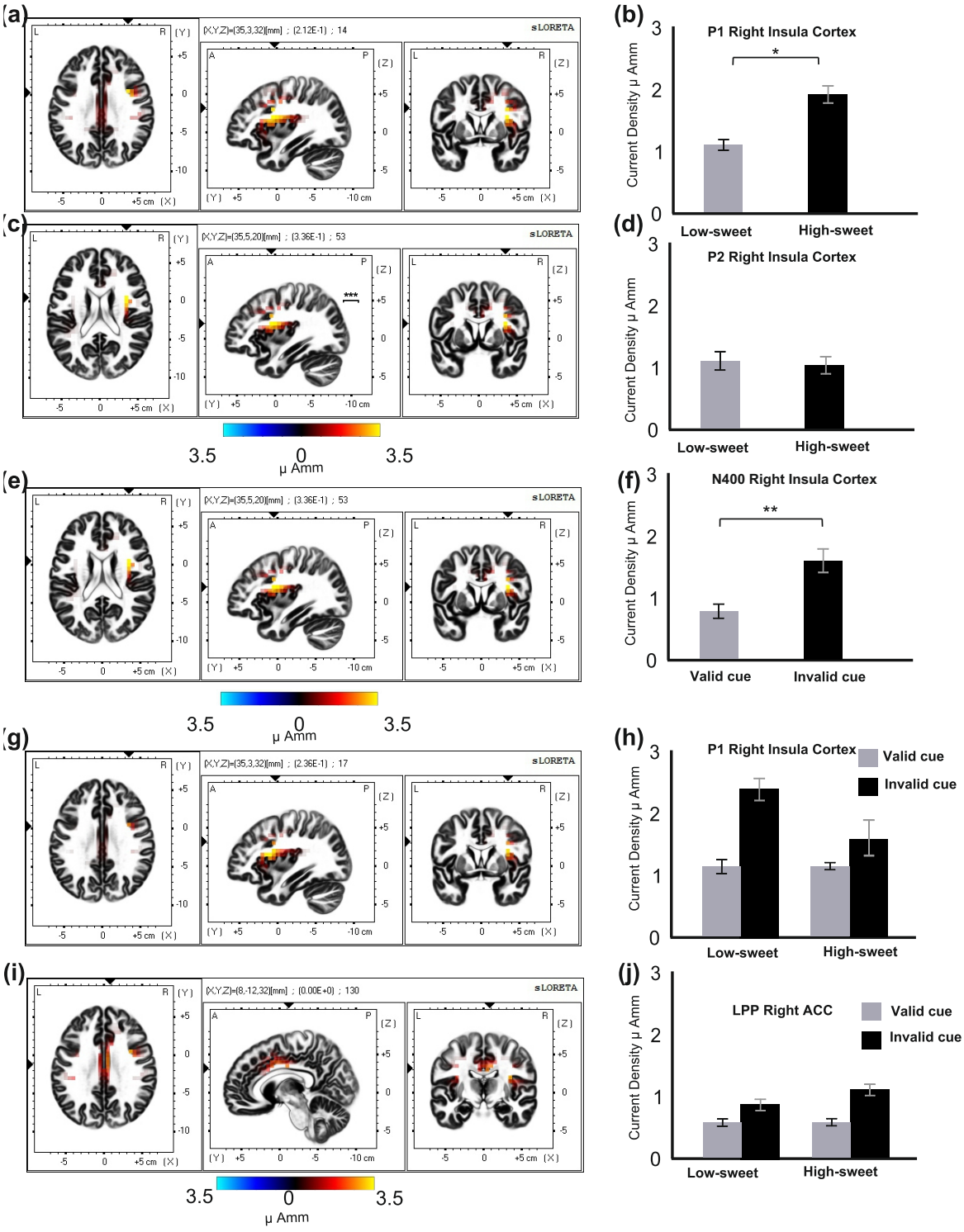
(a)
(b)
P1 Right Insula Cortex
Current Density μ Amm
Low-sweet
High-sweet
(c)
(d)
P2 Right Insula Cortex
3.5
0
3.5
μ Amm
(e)
(f)
N400 Right Insula Cortex
Valid cue
Invalid cue
(g)
(h)
P1 Right Insula Cortex
Valid cue
Invalid cue
(i)
(j)
LPP Right ACC
sLORETA

Fig. 5.6. sLORETA imaging results displaying maximum current density at each ERP latency for the grand mean results, with colour bars representing current density (μ Amm) and bar charts showing the current density for each condition, with error bars indicating standard error. (a) sLORETA image showing the maximum current density at the P1 taste latency (80 – 120 ms) located in the right insula cortex. (b) Bar chart showing the mean current density at this location and latency for each taste condition. (c) sLORETA image showing the maximum current density at the P2 taste latency located in the right insula cortex. (d) Bar chart showing the mean current density at this location and latency for each taste condition. (e) sLORETA image showing the maximum current density at the N400 expectancy latency located in the right insula cortex. (f) Bar chart showing the mean current density at this location and latency for each expectancy (valid/invalid cue) condition. (g) sLORETA image showing the maximum current density at the P1 taste × expectancy latency located in the right insula cortex. (h) Bar chart showing the mean current density at this location and latency for each taste and expectancy condition. (i) sLORETA image showing the maximum current density at the taste × expectancy LPP latency located in the right anterior cingulate cortex. (j) Bar chart showing the mean current density at this location and latency for each taste and expectancy condition. Asterisks indicate significant differences: * $p < .05$; ** $p < .01$.

Note – the sLORETA images show the MNI coordinates for each effect, these have been converted to Taliarach coordinates in the results section (Brett et al., 2002).

5.4.5. ERD/S analysis

As Figure 5.7 illustrates, distinct differences in ERD/S were observed for taste, expectancy and taste × expectancy interactions. The principal effects are summarised below for each factor in relation to theta-, alpha- and beta-band oscillations.

Taste

From Figure 5.7 (a) it can be seen that both tastes induced an early (0.2 – 0.6 s) beta-band (13 – 30 Hz) ERD response and a continuous (0.5 – 2.5 s) theta-band (4 – 7 Hz) ERD response, both of which showed no differences between the taste conditions (*p*s > .148). An effect of taste did, however, emerge on beta-band oscillations (13 – 30 Hz) between 2.2 – 2.5 s across the left fronto-central region (Fc1, Fc5, C1, C5, T7; F (1, 15) = 5.47, p = .034, ES = .27). From Figure 5.7 (b) it can be seen that both tastes induced beta-band ERD, but this effect was greater for high-sweet taste (M = -0.08, SE = 0.03) than for low-sweet taste (M = -0.03, SE = 0.03).

Expectancy

From Figure 5.7 (c) it can be seen that both expected and unexpected conditions evoked early theta-band ERD responses. A significant effect emerged within the between 1 – 1.5 s in the left-frontal region (F7, Fc5, Fc1, C1, C3), F (1, 15) = 7.28, p = .017, ES = .33. As Figure 5.7 (d) depicts, the validly cued condition evoked greater theta-band ERD (M = -.1.33, SE = 0.03) in this region than the invalidly cued condition (M = -0.07, SE = 0.03).

Taste × expectancy interactions

Taste × expectancy interactions were observed in alpha-band (7 – 13 Hz) oscillations in the right-frontal region (Afz, Af4, Af8, F2) between 0 – 0.5 s [Figures 5.7 (e, f)], and in the right central-parietal region (C2, C6, T8, Tp8, Cp2, Cp4, Cp6, P4, P6, P10) between 1.8 – 2.2 s [Figures 5.7 (e, g)].

In the right-frontal region, a significant taste × expectancy interaction emerged F (1, 15) =10.67, p = .005, ES = .42. Specifically, invalidly cued low-sweet tastes produced a similar alpha-band ERD (M = -0.21, SE = 0.06) to validly cued high-sweet tastes (M = -0.13, SE = 0.03). These effects were both greater than the

ERD evoked by validly cued low-sweet tastes (M = -0.07, SE = 0.05) or invalidly cued high-sweet tastes (M = -0.01, SE = 0.06).

In the right central region, the reverse effect was evident, F (1, 15) = 6.58, p = .022, ES = .30. In this case, validly cued low-sweet tastes (M = -0.07, SE = 0.05) and invalidly cued high-sweet tastes (M = -0.08, SE = 0.05) showed similar alpha-band ERD power. Invalidly cued low-sweet taste (M = 0.02, SE = 0.05) and validly cued high-sweet taste (M = 0.07, SE = 0.06) showed little change or slight alpha-band ERS, respectively.

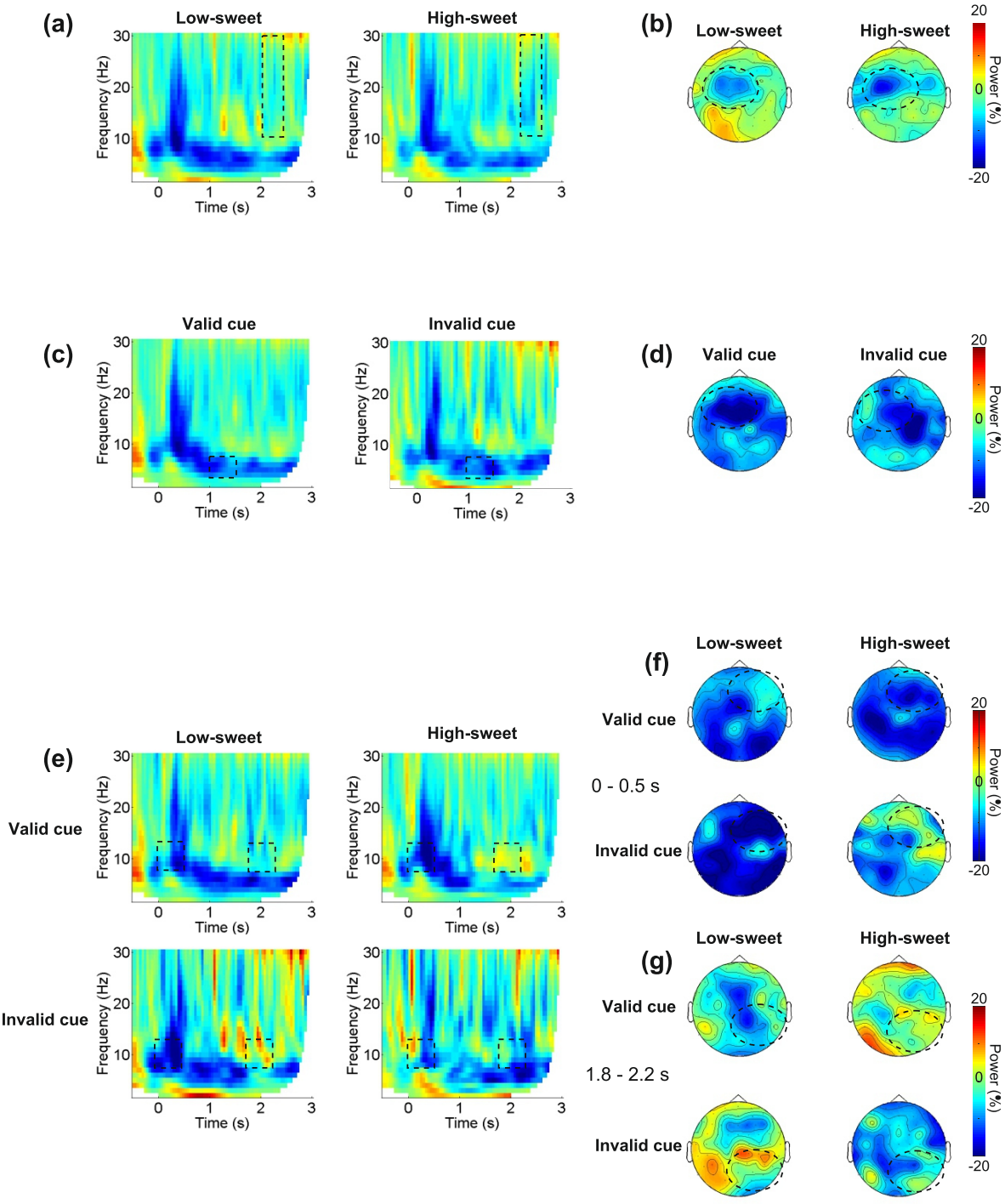
(a)
Low-sweet
High-sweet
Frequency (Hz)
Time (s)
(b)
Low-sweet
High-sweet
Power (%)
(c)
Valid cue
Invalid cue
(d)
Valid cue
Invalid cue
(e)
Low-sweet
High-sweet
Valid cue
Invalid cue
(f)
Low-sweet
High-sweet
Valid cue
0 - 0.5 s
Invalid cue
(g)
Low-sweet
High-sweet
Valid cue
1.8 - 2.2 s
Invalid cue

Fig. 5.7. Time-frequency spectrographs (TFR plots) across all electrodes depicting the power (%) and frequency of oscillations with significant effects indicated by boxes, and topographic head plots for each effect observed in the ERD/S analysis. (a) TFR plots indicating the effects of taste on beta-band oscillations (2.2 – 2.5 s). (b) Topographic headplots of the effects of taste beta-band oscillations. (c) TFR plots indicating the effects of expectancy (valid/invalid cue) on theta-band oscillations (1.0 – 1.5 s). (d) Topographic headplots of the effects of expectancy on theta-band oscillations. (e) TFR plots indicating the interaction effects of taste × expectancy on alpha-band oscillations (0 – 0.5 s; 1.8 – 2.2 s). (f) Topographic headplots of the effects of taste × expectancy on alpha-band oscillations between 0 – 0.5 and (g) between 1.8 – 2.2 s.

5.5. Discussion

In this chapter we investigated the influence of expectations on the temporal, source-localised and oscillatory processing of tastes. EEG data across all electrodes, time points and various frequency bands were evaluated relative to the onset of high- and low-sweet tastes under validly cued and invalidly cued conditions. Behaviourally, expectancy influenced subjective intensity ratings. Articulated gERPs were observed and intensity-dependent P1 differences were found and source-localised to the right insula cortex (PGC). Expectancy ERPs showed N400 amplitude differences in the left- and right-frontal regions, and taste-expectancy interactions were observed in early (P1) and late (LPP) ERP components. Oscillatory data indicated that taste affected left fronto-central beta-band desynchronizations. Expectancy was shown to affect left fronto-central theta-band oscillations, while taste-expectancy interactions influenced alpha-band desynchronizations in the right-frontal and right-parietal regions. These findings suggest that expectancies affect the processing of gustatory stimuli at an early perceptual level and support conclusions drawn from earlier fMRI investigations (e.g., Nitschke et al., 2006; Sarinopoulos et al., 2006; Woods et al., 2011).

Taste

Behaviourally, high-sweet tastes were rated as more intense than low-sweet tastes. However, although the high-sweet solution was initially rated as more pleasant than low-sweet, average pleasantness ratings declined across testing, resulting in both tastes rated as similarly unpleasant during the EEG experiment. That the P1 amplitude of the gERP was shown to be increased for the high-sweet compared with the low-sweet tastes supports our interpretations of the findings in Chapters Three and Four of potentially taste intensity-dependent early ERP deflections (see also Hummel et al., 2010b; Ohla et al., 2010). Moreover, we were able to discern that P1 effects were localised to the right insula cortex in the PGC, which showed greater current densities for high-sweet tastes, corroborating earlier reports of intensity dependent changes in primary gustatory regions (e.g., Ohla et al., 2010).

Taste dependent differences were also observed within beta-band oscillations: greater desynchronization was observed for high-sweet taste, supporting our previous

finding in Chapter Three of increased beta-desynchronization for stronger intensities of different tastants. Beta-band ERD is commonly associated with activations in the sensorimotor cortex (e.g., Paradiso et al., 2004), although it has been reported for auditory (e.g., Makinen et al., 2004), somatosensory (Neuper et al., 2001; Pomper et al., 2012) and olfactory modalities (Miyanari et al., 2006). Moreover, greater beta-band ERD has been reported for strong, compared with weak painful (Pomper et al., 2012) and olfactory stimuli (Miyanari et al., 2006), suggesting that beta-band ERD may vary as a function of stimulus intensity across a number of sensory modalities.

Expectancy

We observed lateralised ERP differences in response to expected and unexpected stimuli. In the left-frontal region, enhanced N400 amplitude was observed in response to validly cued stimuli, an unexpected finding, whereas in the right-frontal region a reduced N400 was seen in response to validly cued stimuli. The N400 latency was source-localised to the right insula region in the PGC where activations to validly cued stimuli were reduced compared with those that were invalidly cued. The N400 ERP is commonly associated with unexpected outcomes in language processing (see Kutas & Federmeier, 2011, for a review), with a right-hemisphere bias that is thought to be related to its involvement in meaning comprehension (e.g., Federmeier, 2007). However, the N400 has been observed for incongruencies within audition (e.g., Painter & Koelsch, 2011), vision (e.g., Proverbio & Riva, 2009) and olfaction (Kowalewski & Murphy, 2012). For the first time, this effect can be now extended to primary gustatory processing. However, the left-lateralised effect for expected stimuli requires further examination.

Theta-band ERD was enhanced when tastes were validly cued compared with the invalidly cued condition. Theta-ERD was linked with habituation to tastes in Chapter Three (see also Tóth et al., 2004). Therefore, the current results may represent increased habituation to the more frequently presented (i.e., 70 % of trials) expected taste condition. We also noted early beta-band ERD responses in both expected and unexpected conditions. Early beta-band activity is related to anticipatory processes (Donner et al., 2007; Roelfsema et al., 1997; Schoffelen et al., 2005), and desynchronizations have been observed when an event has been predicted

(e.g., Fujioka et al., 2012). Thus, the current results may reflect the anticipation of stimulus presentation.

Taste × expectancy interactions

The behavioural results indicated that intensity ratings for high-sweet tastes decreased when participants were expecting a low-sweet taste, while intensity ratings for low-sweet tastes increased when a high-sweet taste was expected; supporting the findings of previous studies (Nitschke et al., 2006; Sarinopoulos et al., 2006; Woods et al., 2011). Importantly, taste expectancy interactions occurred in early ERP processing. P1 amplitudes for invalidly cued low-sweet tastes were decreased to a level similar to that for high-sweet tastes and P1 amplitudes for invalidly cued high-sweet taste increased to a level similar to that for low-sweet tastes. These data indicate that intensity expectancies influence sweet taste processing at an early perceptual level. These results strengthen findings reported in fMRI investigations (e.g., Nitschke et al., 2006; Sarinopoulos et al., 2006; Woods et al., 2011) and suggest that expectancies not only alter processing in primary sensory regions but also affect early sensory processing stages. In the later evaluative ERP processing phase, expecting a high-sweet taste but receiving a low-sweet taste evoked the greatest amplitude of all conditions, suggesting that this may be the most salient of incongruent outcomes, as such late ERP effects can be attributed arousing events (e.g., Hajcak et al., 2010).

Taste-expectancy interactions also occurred in alpha-band oscillations, which were associated with the processing of sweet tastes in Chapters Three and Four. Alpha-band ERD for low-sweet tastes was increased when participants were expecting a high-sweet taste. Conversely, alpha-band ERD for high-sweet tastes was decreased when participants were expecting a low-sweet taste. Thus, alpha-band responses for unexpected tastes appear to assimilate to the responses for the expected taste, similar to our ERP findings and the PGC activations observed the fMRI investigations (e.g., Nitschke et al., 2006; Sarinopoulos et al., 2006).

Limitations

We recognise that the pleasantness evaluations did not differ consistently between tastes during the experiment, despite clear differences in initial ratings. Pleasantness

ratings were also shown to decline over the course of the experiment - despite measures taken to reduce habituation and adaptation, while intensity ratings for the two solutions remained stable and distinct. Thus, the current data cannot provide any clear inference about the influence of the hedonic value of the stimuli. Our strategy of collecting data in a single session was designed to reduce any EEG noise that may otherwise have been incurred with multiple test days and potential differences in electrode sites. The consequences for stabilised hedonic responses for any EEG study measuring responses to repeated taste stimuli should be accounted for in future investigations. Nevertheless, the present study has reliably determined that expectancy modifies perceived intensity of intrinsically pleasant, sweet stimuli and evokes cue-dependent changes in cortical activation. Moreover, since our expectancy cues were presented solely in terms of relative sweetness, it is likely that they may not have had a direct impact upon anticipated or actual palatability evaluation, and that our findings specifically reflect alterations in the processing of intensity (Okamoto & Dan, 2013).

Although not necessarily a specific limitation of the current study, another factor that may be considered in future studies is the nature of cortical activation related to the anticipatory cues themselves. Evidence from the fMRI studies described earlier suggests that regional changes while viewing a cue may determine whether, to what extent, and in which direction the cues influence the perception and evaluation of taste stimuli (Sarinopoulus et al., 2006; Okamoto & Dan, 2013).

Conclusions

In sum, we were able to observe fully articulated gERPs, likely resulting from an increase in trial repeats and a decrease in the overall testing duration. The combination of these factors led to a reduction in trials lost through the removal of noisy data and an increase in data available for analysis. It is also possible, however, that the addition of a cognitive component (i.e., expectancy) led to attentional affects that may have contributed to the ERP. Although not possible with the current ERP data (as it was collected in a single session and was not pre-processed or analysed on a trial by trial basis), one way to tease apart these effects in future may be to examine whether an average of 20 trials (as observed in Chapter One) of this paradigm results in an articulated ERP response. If this were to occur, this would indicate that it was

the addition of a cognitive component - rather than an increase of retained trials - that led to the production of an ERP.

Within the gERP data, an intensity dependent P1 component and source localised PGC intensity effects were observed, further highlighting the role of early ERPs and the PGC in taste intensity processing (e.g., Ohla et al., 2010; Sadacca et al., 2012). We were also able to demonstrate that the N400 ERP component, observed for incongruencies within other sensory modalities, can be determined in response to unexpected taste stimuli and are generated from within PGC areas. Importantly, our data demonstrate that prior expectations can not only modify subjective intensity ratings of taste, but also affect early sensory representations as measured using the fine temporal resolution enabled by analysis of EEG responses. These data strengthen conclusions drawn from fMRI studies that report taste-expectation interactions in primary gustatory cortices (e.g., Nitschke et al., 2006; Sarinopoulos et al., 2006; Woods et al., 2011) and show that early sensory representations can be modified by top-down information. Moreover, the current study extends the analysis of expectation of the processing of sweet taste intensity, and provides a first demonstration of clear bi-directional effects of expectancy cues on cortical activation by pleasant, sweet tastants. Taken together, these findings support the assimilation effects predicted by the Assimilation-Contrast Model of expectation (Heider, 1944; Sherif & Hovland, 1961) in that taste intensity perception and concomitant neural processing tend to shift toward responses that would be engendered were the actual taste stimulus to match expectation. Moreover, in contrast to a previous report (Woods et al., 2011), assimilation was apparent irrespective of the direction of the discrepancy between expected and actual level of sweetness. This phenomenon has important implications for understanding the role of extrinsic cues involved in food choice and experience (Davidenko et al., 2015).

Chapter Six: General Discussion

6.1. Review of Thesis Aims

The overarching aim of this thesis was to examine the central processing of tastes and the influence of physiological and psychological factors on this mechanism. To do this, a number of objectives were outlined in Chapter One (section 1.9). These included devising a taste stimulus set that can measure the separate characteristics of taste quality, intensity and hedonicity; the development of a computer controlled gustometer device; exploration of the coding of taste characteristics using EEG; understanding the influence of hunger and satiety on the EEG processing of tastes and to investigate the effects of expectancy on taste coding. The following discussion will address each of these objectives and discuss the extent to which these were met, as well as the limitations and implications of the findings.

6.2. The Development of the Taste Stimulus Set

As discussed in Chapter one (section 1.6) and Chapter Three, investigations into the neural coding of taste quality, intensity and hedonicity often show an overlap in the processing mechanisms identified for for each of these characteristics, making it difficult to separate the processing of one taste attribute from another (e.g., Small et al., 2001b). This difficulty can be largely ascribed to the fact that behaviourally, these factors are very intertwined and researchers have tended to measure responses to one taste characteristic without adequately controlling for another (e.g., Franken et al., 2011; Grabenhorst & Rolls, 2008; Hummel et al., 2010). Given this, it is vital for researchers to take careful consideration of the stimulus set when examining responses to tastes. Currently, there are no standardised stimuli sets for measuring responses to gustatory stimulation.

In order to determine an appropriate range of stimuli for the investigations reported in this thesis, we conducted a series of preliminary tests whereby 50 participants rated 81 bitter, sweet and salt tastants for their quality, intensity and pleasantness. From this, we were able to ascertain a number of things. Firstly, certain lower-concentrations of tastes were unable to be consistently identified for their taste

quality and thus are not suitable for gustatory investigations. Secondly, pleasantness ratings for bitter and salt decreased with increasing concentrations, thus the interactions between these factors should be considered in all taste investigations. For instance, if examining intensity responses to increases in NaCl concentrations, one should be mindful to select tastes that do not also show decreases in pleasantness ratings. Thirdly, ratings for sweet tastes did not show interactions between intensity and pleasantness, rather, there was little change in rated pleasantness across concentrations. Moskowitz (1971, 1979, 1982) also reported no interactions between sweetness and pleasantness and these results suggest that it is not viable to measure taste hedonicity as a psychophysical function of intensity. Lastly, by comparing the ratings of different tastants, we were able to determine a stimulus set that could distinguish between different tastant characteristics.

For Chapter Three, we required tastes that could be formed into groups that were easily recognised for their taste qualities (sweet, salt, bitter); were statistically distinguishable in their ratings for taste intensity (weak, medium strong) and differed in their hedonic value (pleasant, unpleasant). We were able to successfully meet these criteria by only selecting taste that were reliably recognised and differentiated for their taste quality and that statistically differed in their ratings of intensity and valence.

When utilising this stimulus set for the EEG testing in Chapter Three, we found that the differences reported in the intensity and pleasantness ratings during the preliminary study were replicated when measured prior to the EEG study. This corroborates the reliability of these selected tastes for the discrimination of quality, intensity and hedonicity. Furthermore, this selection of tastants allowed us to successfully discriminate neural responses to intensity and hedonicity.

For Chapter Four, we measured responses to tastants under hungry and sated conditions. We were particularly interested in whether reported effects of hunger and satiety on taste perception were specific to pleasant, nutrient rich tastes, or were part of a broader mechanism affecting tastes in general. We consequently selected a sucrose and QHCl concentration as they differed in valence but not subjective intensity in the preliminary investigation. As we had reduced the stimuli set compared with Chapter Three, we were able to increase the quantity of repetitions for 40 per tastant; compared with 30 in the previous chapter.

In Chapter Four, we also recorded ratings of intensity, pleasantness and arousal over the course of the experiment. Ratings of pleasantness were consistent with our preliminary investigation in that bitter and sweet tastes, as expected, showed significant differences in pleasantness ratings. However, contrary to our preliminary study, intensity ratings did show significant differences, with bitter rated as more intense than the sweet stimulus. Arousal ratings showed the same effects, and were strongly correlated with intensity ratings. It is suspected that reducing the stimulus set to just three samples resulted in an increase in the discrimination between the ratings of intensity. Although we were unable to control for this effect, we were, however, able to account for it by including the online ratings and subsequently, some of our results could be attributed to effects of taste intensity (and/or arousal).

In Chapter Five, we examined the influence of expectancy on the processing of sweetness. Due to our preliminary study, we were aware that pleasantness ratings were unlikely to change with concentration so we examined the effects of cue-elicited expectation on responses to taste intensity. We selected two concentrations of sucrose that significantly differed in intensity ratings in the preliminary investigation, but were not so different in quality or pleasantness that could result in expectancy-contrast effects (e.g., Wilson and Klaaren, 1992). As we employed just two taste conditions, we were able to increase the quantity of repetitions to 50 per tastant.

In this study, we obtained baseline pleasantness and intensity ratings of the tastants in conjunction with extracting online ratings during the course of the experiment. The baseline ratings showed significant differences in rated intensity, consistent with our preliminary study. However, the baseline ratings also showed differences in rated pleasantness between the sucrose solutions, which did not fit with the preliminary findings. Again, as with Chapter Four, the further reductions in stimuli comparisons likely resulted in an increase in discrimination between them. However, contrary to the baseline ratings, the averaged scores over the course of the experiment showed that the pleasantness ratings for the two sucrose concentrations no longer showed differences and, in fact, were both rated as equally unpleasant. This was not an issue for this analysis as it was taste intensity outcomes that were examined and these were retained. However, potential changes in the pleasantness

evaluations of tastes following repeated stimulation should be considered in future investigations.

Limitations

The selections of the taste stimuli were carefully determined based on statistical data from an extensive preliminary study. This method was successful for the study reported in Chapter Three, as the ratings gained in the experiment were consistent with those obtained during the preliminary study. Thus, we were able to measure the separate coding mechanisms for different taste characteristics. However, the employment of such an extensive stimulus set in this chapter meant the number of stimuli repetitions was constrained and as a result, no articulated ERP peaks were observed. Therefore, while we observed ratings consistent with the preliminary investigation, the resulting consequences for the EEG data were unfavourable. Moreover, the ratings taken from the EEG participants were sought just before the EEG study, with no online ratings recorded over the course of the experiment. Thus, it cannot be determined if the taste ratings were maintained over the course of the experiment. While not taking ratings during the course of the experiment may have reduced testing time, this has limited us in our inferences from the data.

For Chapters Four and Five, we decreased the stimulus set, increased the number of repetitions and recorded online ratings of the tastants. However, this method resulted in a further limitation in that our data showed that the fewer tastants employed, the bigger the differences in ratings between them. Therefore, the ratings gained from the EEG investigations in those Chapters did not always match the data from the preliminary study. Fortunately, we were able to account for these differences in our interpretation of the data as we had recorded the ratings of the tastes over the course of the experiments. However, for future studies it is important to consider that ratings gained from piloting tastants may not always match those reported during testing and the differences may be accounted for, in part at least, by the quantity of stimuli employed.

Although not necessarily a limitation for our investigation, a further observation is that the ratings of repeated taste stimuli can change over the course of the experiment. As observed in Chapter Five, 50 repetitions of each sweet tastant resulted in a steady decrease of pleasantness ratings, to the extent that the mean

ratings over the course of the experiment showed that both sweet tastes were deemed unpleasant. Fortunately, the differences in intensity ratings were maintained and were consistent with our preliminary investigation. This meant that we were able to measure the changes in reported intensity ratings along with the concomitant neural responses to cue-elicited expectancy as intended. However, this may be a limitation for taste processing studies in general. It is recommended that to measure EEG responses to taste, many repetitions are required (e.g., Mizoguchi et al., 2002). However, our findings indicate that repeated stimulation with sweet tastes at least, results in a change in the hedonic value of that tastant and future studies should account for this shift in responses.

Implications

There are a number of crucial implications of these findings that are relevant for future investigations of taste processing. Firstly, there exists no standardised stimuli set for measuring responses to tastes and little consistency between studies, therefore it is important to conduct preliminary testing and select tastes that show significant differences in ratings. This means that when making inferences about data, we can be certain that they are observing responses to the correct taste characteristic. For instance, that responses to taste intensity are observed and not responses to hedonic factors, which can be highly correlated particularly for unpleasant tastes (see Chapter Two, section 2.3).

Our findings indicate that 50 stimuli repetitions and a testing duration of ~1 hour can result in fully articulated ERP components, compared with fewer trials and increased testing durations. However, as discussed, repeated stimulation of sweet tastes resulted in decreased ratings of sweet pleasantness. Had we not recorded baseline as well as online ratings of these tastant these changes may have been overlooked. Thus, it is important to record both ratings of tastants on the day of testing, as well as over the course of the trials to monitor any changes that might occur. Furthermore, these differences in ratings may be avoided by conducting EEG studies over several sessions (e.g., Mizoguchi et al., 2002). Although this may be costly and taxing for researchers and participants, it may serve to decrease repetitive stimulation and thus avoid any changes in ratings. However, this method is controversial as recording EEG over several sessions may impede the quality of the

EEG data. Differences in EEG recordings can vary quite extensively for trivial reasons such as differences in the positioning of the cap, or hair, resulting in different electrode sites across sessions (Luck, 2005). Therefore, single sessions are usually preferred when measuring the same variable. As such, the need for a consistent quality in the EEG data must be weighed against the need for stabilised behavioural measures.

6.3. The gustometer

In order to measure EEG responses to gustatory stimulation a gustometer system and accompanying program is required that meets a number of prerequisites. This is discussed in full in Chapter Two (section 2.4.1), but to summarise; the system must have excellent temporal precision in that it is able to present taste stimuli that reach the mouth at the same time as a trigger to the EEG system (Ohla, 2011). The tastes must also be delivered with steep rise times (< 50 ms) so that early latency EEG changes in can be observed (Evans et al., 1993; Ohla, 2011) and the system must provide rinses between tastes and long ISIs in order to avoid habituation and adaptation effects (Mizoguchi et al., 2002).

In addition to the published requirements of such a system (e.g., Evans et al., 1993; Mizoguchi et al., 2002; Ohla, 2011), we aimed to develop a gustometer and program that also controlled for a number of other factors. We required a program that included a component whereby manual initiation of trials was possible so that we could ensure that the trials were not synchronous with swallowing motions that accompany tasting. We also required a program that allowed for the indication of unsuccessful trials (that may incur as a result of a failure of the system to pump the tastants) within the EEG and behavioural data. Lastly, we needed to ensure the any electrostatic charge generated from fluids passing through Teflon tubing was discharged prior to reaching the participants so that it did not interfere with the electrical data recording, or produce an unsafe environment for the participant.

We were able to successfully establish a gustometer with a custom built program that met all of the above requirements. The gustometer had excellent temporal precision with a rise time within 20 ms. The custom built software allowed us to manually initiate trials when we could be certain that participants were not swallowing, thus allowing for a reduction of noise within the EEG data. Further, the

program allowed for the insertion of indicators in the EEG data when pump failure occurred and triggered the termination of the trial sequence upon pump failure. Lastly, we were able to successfully discharge the static electricity generated by the moving fluids before it reached the participant. Therefore, our data was free of static interference and the system was safe for use.

Limitations

The development of the gustometer and accompanying program was an intensive and time-consuming task. It was almost two years before EEG testing could take place, which subsequently limited the number of investigations that could be conducted within the time-limit of the Doctoral degree. However, the knowledge and gained from this could greatly inform future investigations on the development of an economically viable gustometer and specific techniques to reduce the signal-to-noise ratio in gustatory EEG.

While we were able to establish a rise time of < 20 ms, this was calculated prior to the investigations and was not recorded during real-time tasting. Intra-oral measurements were not possible in the current studies, given the size of the water sensor (45 mm x 70 mm). Future designs could look to condense such a sensor into a compact mechanism that can be placed intra-orally for a more accurate, online assessment of rise-time.

A factor which may be considered a limitation is that occasionally the pumps failed to work and this required a restart of the system of the system. This fault occurred in an average of 17.7 % of trials in the study reported in Chapter Three, 14.3 % of trials in Chapter Four and 17.4 % in Chapter Five. While this factor cannot be controlled, it can be accounted for when determining the number of stimuli repeats. As a result, the number of trials was increased for each of the investigations reported in this thesis.

Implications

We were able to successfully develop a gustometer system that fit the technical requirements of EEG investigations (e.g., Evans et al., 1993; Mizoguchi et al, 2002; Ohla, 2011). Further to this, we were able to develop new methods to control for the

electrostatic charge generated from moving fluids in the gustometer system, and provide a technique to limit noise generated by muscle movements that accompany swallowing. These techniques are novel to gustatory EEG and provide crucial solutions to reducing noise and increasing the quality of the EEG data. Combined with an ample quantity of stimuli repetitions and a limited testing duration, this system can deliver tastants in a way that the quantification of peak ERP components is achievable. Therefore, our techniques can greatly inform future research in this area. Moreover, future studies could build upon these methods, allowing for further improvements to gustatory EEG. For instance, algorithms could be developed in order that the initiation of trials could be performed in the absence of EMG activity, without the need for manual initiation from the researcher, and water sensor devices could be designed to be compact enough to be employed intra-orally.

6.4. Dissociating the Gustatory Coding of Quality, Intensity and Hedonic Value

As discussed in Chapter One (section 1.6) and Chapter Three, dissociating the processing mechanisms involved in the coding of taste quality, intensity and hedonicity is challenging given the overwhelming associations between these characteristics (e.g., Pfaffmann, 1980; Small et al., 2001b). In Chapter Three we employed a carefully devised taste stimulus set that could reliably distinguish between these tastes features and explored ERPs, the source-localisation of ERPs and neural oscillations in responses to tastes of varying quality, intensity and hedonicity.

The key findings from this investigation indicated that taste intensity was represented by alpha- and beta-band ERD, which both increased for stronger taste intensities and showed little change in response to weaker tastes. Taste hedonicity processing was characterised by an increase in left-lateralised alpha-band ERD in response to pleasant tastes (compared with unpleasant and neutral tastes), and by increases in activations from the right inferior parietal lobule for pleasant tastes. The ERD/S data are consistent with increasing evidence that left-lateralised alpha-ERD may be particularly responsive to pleasant stimuli (e.g., Basar et al., 2012; Eckhorn et al., 1988; Engel et al., 2001; Kayser et al., 2012; Nicolelis et al., 1995; Laurent & Davidowitz, 1994), and that such neural oscillations may be necessary in order for sensory discriminations to take place (e.g., Nusser et al., 2001; Stopfer et al., 1997).

Moreover, the findings suggest that further analysis of the role of the inferior parietal lobule in taste processing should be considered.

While ERP analyses were able to indicate that taste intensity was processed in early processing epochs (60 – 140 ms) and hedonicity at later epochs (600 – 1500 ms), no articulated ERP responses were obtained and the observed amplitude differences did not fit the psychophysical attributes of the taste. For example, ERP responses to strong and weak tastes differed from neutral and medium, and responses to hedonically positive and negative tastants differed from neutral but not each other. It is possible that the ERP responses were confounded by arousal, however, it is also likely that the limited stimuli repeats and trials retained after pre-processing affected the ability to discern some ERP amplitude changes that may have been observable had there been more available data. Thus, ERD/S analysis may lend itself better to the discrimination of taste intensity and hedonic processing mechanisms, particularly in studies where repeated stimulation is an issue.

Neither ERP, source-localisation nor ERD/S were able to discern specific differences in the processing of taste quality other than greater responses observed for flavoured tastes compared with water; effects that are more consistent with data usually attributed to arousal or hedonicity (Balconi & Mazza, 2009; Davidson & Henriques, 2000; Hajcak et al., 2010b; Waldstein et al., 2010b). High-density fMRI scanning and intracranial recordings may be better suited to examining taste quality coding (Crouzet et al., 2015).

Overall, we were able to conclude that when applying a stimulus set that could reliably differentiate between taste quality, intensity and hedonic value; ERD/S and source-localisation analysis, in particular, may lend themselves well to the dissociation of neural responses to these taste characteristics. Specifically, the discrimination of taste intensity may be mediated by alpha-and beta-band oscillations, and hedonicity processing may rely on left lateralised alpha-ERD and activations within the inferior parietal lobule.

Limitations

In order that the analysis could be conducted within a single session, the extensive stimulus set and ISI had to be offset by limiting the number of repetitions within

each stimulus category. However, the testing duration was extensive, which resulted in substantial noise within the EEG data and a considerable loss of trials. Consequently, we were unable to discern articulated gERP peaks. Thus, the following investigations aimed to reduce the quantity of tastants in the stimuli set and increase stimulus repetitions, which was shown to be an effectual solution in Chapter Five. However, the absence of typical gERP peaks when averaged across subjects is common within the literature (e.g., Crouzet et al., 2015; Prescott, 1994; Singh et al., 2011). Thus, while it is preferable to obtain these articulated results, an absence of them does not prohibit the effective analysis of the data as shown within these studies and our own results reported here, although it may be prudent to take caution when interpreting the results.

One factor that may be considered a limitation for all of the studies reported here is the source-localisation analysis. As discussed in Chapter two (section 2.5.4), source-localisation relies on a solution to the inverse problem. However, the reality of how the signal was generated is not known. It is therefore up to the user to decide whether or not the constraints used in a given inverse solution are physiologically plausible (Michel et al., 2004). However, sLORETA has received considerable validation from studies combining this software with other more established localisation methods, such as fMRI (Mulert et al., 2004; Vitacco et al., 2002), structural MRI (Worrell et al., 2000) and intracranial recordings (Zumsteg et al., 2006a; Zumsteg et al., 2006c).

Implications

The current data go someway to characterise the temporal and oscillatory dynamics of the processing of taste quality, intensity and hedonicity. In particular, our data suggest that oscillatory processes may be crucial in discriminating tastes for their intensity and pleasantness characteristics. The oscillatory dynamics of human gustatory processes are rarely explored (Fox & Davidson, 1986; Morinushi et al., 2000; Tóth et al., 2004), in favour of MRI and ERP methodologies. However, there is increasing evidence to suggest that neural oscillations play a vital role in sensory processes (Klimesch et al., 1996; Nusser et al., 2001; Palva et al., 2005; Pfurtscheller & Lopes da Silva, 1999; Pfurtscheller et al., 1994; Sausing et al., 2005; Stopfer et al., 1997). Our results extend these findings to include gustatory coding and highlight

the importance of oscillatory analysis in understanding the processes involved in discriminating taste characteristics.

Future studies may obtain more reliable ERP data by conducting this study over several sessions. Although this is controversial within EEG investigations (Luck, 2005), increasing the number of stimuli repetitions and reducing study durations (by using repeated sessions) is likely to result in a reduction of noise and, by extension, data loss. By increasing the amount of data available, the likelihood of achieving more articulated ERPs and more reliable data is enhanced and would ultimately improve the interpretation of the findings. Improving upon this method may then open the door for a more detailed examination of these processes. For example, it would be interesting to examine the role of individual differences in the processing of gustatory information. Studies have suggested that factors such as BMI (Drewnowski et al., 1985), gender (Yamaguchi et al., 2002), ethnicity (Mennella et al., 2005) and thermal taster status (Hort et al., 2016) impact up the perception of tastes. Understanding the role of these variables could be particularly informative for research investigating factors associated with food choice and may inform future research and interventions for those with abnormal eating behaviours, for example, the tailoring of dietary advice.

6.5. The Effects of Hunger and Satiety on the Processing of Tastes

Appetite can influence the perception of taste (e.g., Cabanac, 1971) and in Chapter Four we examined the neural underpinnings of this relationship. We measured EEG responses to pleasant (sweet), unpleasant (bitter) and neutral (water) tastes in participants who were either hungry following a 12 hour overnight fast, or sated following the consumption of a liquid meal.

The results of this study indicated that while hunger state did not generally affect the intensity, arousal and pleasantness ratings of the tastes (aside from sweet being rated as less pleasant when sated), it greatly affected the neural processing of tastants. Frontal ERP responses across all tastants, as well as parietal beta-band ERS, were considerably enhanced during hungry states. The ERP findings can be attributed to increased attentional mechanisms employed during the processing of tastes or food-related stimuli when hungry (e.g., Stockburger et al., 2008). In

contrast, however, beta-ERS is suggestive of cortical inhibition and avoidance related measures (Coan & Allen, 2004; Harmon-Jones et al., 2010; Klimesch et al., 1998; Schutter et al., 2007). It is therefore possible that while there was increased attention to tastes under hungry conditions, some mechanisms may be actively supressing the motivation to eat as has been reported previously (see Yoshikawa et al., 2004). However, given the limited knowledge on the oscillatory dynamics of taste processing, such an interpretation is tentative at this time.

Hunger and taste were also shown to interact within the EEG data. Both ERP and ERD/S findings indicated that the interaction was largely a result of the differences in the processing of sweet tastes in hungry and sated conditions. While in general, hunger enhanced ERP responses to tastes; responses to sweet tastes were greater when full, whereas responses to bitter did not change after satiation. These results suggest that increased attention was assigned to the pleasant tastes when hungry, whereas the attention to bitter tastes was not altered by hunger state. Similarly, alpha-band ERD occurred in response to sweet tastes when sated (as occurred in response to sweet tastes in Chapters Three and Five), whereas alpha-ERS occurred in hungry states. As with the beta-ERS, these results are suggestive of cortical inhibition (e.g., Palva et al., 2005). Thus, the alpha-ERD observed for pleasant tastes seems to be actively inhibited during states of hunger. Therefore, while the attention to, and the evaluation of sweet tastes is enhanced when full, the evaluation of pleasantness seems to be supressed following an overnight fast. In Chapter four we speculated that during hunger, the energetic qualities of the sweet taste may take priority over its hedonic value (e.g., Benson, 1977; Sheppard, 1975; Sherratt, Speed & Ruxton, 2003; Strygley & Kingsolver, 1998).

The source-localisation data in this investigation were also interesting. While in Chapters Three and Five activations to tastes occurred mostly within PGC areas; the findings here indicated that it was the limbic areas (associated with the processing of emotional arousal) that showed the greatest magnitude of current density. The more intense and arousing pleasant and unpleasant tastes evoked greater current densities within the cingulate region, compared to those evoked by water. Similar regions were shown to be activated by tastes during the hungry condition in the fMRI study conducted by Hasse et al. (2009). While no source localisation effects specific to hunger state were observed in our investigation, the findings do

suggest that manipulating hunger through fasting or feeding to satiation, does influence where the greatest responses to tastes occur; so that rather than primary sensory regions showing the greatest current densities; regions associated with emotional arousal appear to more active during these states.

Limitations

There may be a confounding influence induced by employing a sweet liquid meal to induce satiety, thus we cannot be certain if any of the EEG effects observed were attributed to a broad fullness effect or whether they would differ as a function of sensory specific satiety were a savoury meal employed (e.g., Rolls et al., 1991). Moreover, the satiation procedure resulted in increased ratings of nausea compared with the fasting condition, and increased ratings of thirst were observed in the hungry condition. These factors may have contributed to the neural responses to the tastants and efforts should be made to avoid these in future, for instance, by reducing the volume of the liquid meal and ensuring the participants are equally well hydrated.

As with Chapter Three, we were unable to observe typical peak ERP components and we again suspect this is related to the quantity of stimuli repetitions employed and thus this was addressed in Chapter Five. As previously mentioned, however, this limitation is common within gustatory EEG investigations (e.g., Crouzet et al., 2015; Prescott, 1994; Singh et al., 2011) and did not impede our ability to discern temporal differences in the processing of tastes during hungry and sated conditions, although we interpret the findings with caution. As discussed for Chapter Three, in order to improve the reliability of the ERP signal, future studies could examine these effects over more than two EEG sessions. Although this may be lengthy and taxing for researchers and participants, and could result in increased attrition rates, this method would reduce individual testing times, allow for increased stimuli repetitions and provide an increase in clean data available for analysis. These factors would likely improve the ERP signal and our ability to make assumptions based on the findings.

As addressed earlier (section 6.1), the ratings of the tastes differed from those gained in the preliminary investigation. However, as we had recorded online ratings

of the tastants, we were able to account for these differences in our interpretation of the results.

Implications

The findings indicate that hunger has a significant effect on the processing of tastes. In particular, the data observed in the sated condition was more compatible with our findings in Chapters Three and Five, suggesting that being hungry modifies how the brain promotes attention to and the evaluation of tastants. As well as highlighting the importance of controlling for possible confounding effects of hunger when examining neural responses to tastes; the results also have some wider implications. Specifically, our results imply that being hungry enhances attention to taste but decreases the importance of hedonic evaluation, particularly in relation to pleasant, nutrient rich tastes. In evolutionary terms, the nutritional value of a food may take precedence over its palatability when an organism requires energy. Such responses may have developed in order that palatability does not take priority when searching for food, promoting survival when food resources are limited (e.g., Benson, 1977; Sheppard, 1975; Sherratt, Speed & Ruxton, 2003; Strygley & Kingsolver, 1998). Additionally, our observations may reflect the operation of 'wanting', in which the incentive motivational value of a stimulus (food *per se*) that matches a specific motivational state (hunger) is distinct from its hedonic impact ('liking'), enabling the pursuit of a goal even in advance of any hedonic experience of it (Berridge, 2004). By contrast, it is a common experience that, after satiation, the hedonic properties of sweet taste can induce further consumption. These possibilities obviously require further investigation.

It would be interesting to determine whether hunger affects the processing of other sensory stimuli, suggesting a general metabolic effect, or whether these results are exclusive to food related stimuli (e.g., Stockburger et al., 2008), related to a specific mechanisms involved in eating behaviours. In future, studies could examine this possibility by employing a non-food condition with which to compare findings to food or taste related stimuli.

Future studies should also examine whether the type of meal employed to induce satiation has an effect on the neural processing of tastants. In particular, it would be interesting to see if satiety induced by a savoury meal affects the

processing of sweet tastes differently to that resulting from the ingestion of a sweet meal as reported here. However, it may be that in general, hunger reduces the significance of the hedonic evaluation of tastants compared with satiety, irrespective of the meal consumed to satiation. Such findings could greatly inform research on how people make food choices and evaluations under these conditions.

Lastly, the current investigation excluded obese and under-weight populations as a result of differences in reported neural responses to hunger and satiety compared with normal and over-weight populations (e.g., DelParigi, 2002; Gautier, 2000; Wang, 2009). Future studies could benefit from the comparison of different BMI populations in their EEG responses to tastants under hungry and sated conditions. Such research could determine if there are any differences in the temporal taste processing mechanisms that may account for specific differences in eating behaviours between these groups.

6.6. The Effects of Expectancy on Taste Processing

Taste expectations have been shown to affect both sensory and hedonic ratings of tastants (e.g., Du Bose et al. 1980; Stillman et al., 2012). In Chapter Six, we explored the temporal underpinnings of this phenomenon to determine how expectancy affects the processing of tastes. Participants were validly or invalidly cued to expect either a high- or low-concentration of a sweet taste and asked to rate the expected and actual intensity and pleasantness of the tastes while EEG was recorded.

The principal findings indicated that both behavioural and neural responses were modified by the cue-elicited expectation. Behaviourally, intensity ratings for the low-sweet tastes increased when the participants were expecting a high-sweet taste, while intensity ratings for the high-sweet taste decreased when participants were expecting a low sweet taste, in line with the assimilation affects predicted by the Assimilation-Contrast Model (Heider, 1944; Sherif & Hovland, 1961; Wilson & Klaren, 1992). ERP and ERD findings showed that responses for the invalidly cued tastes assimilated to those that were produced by the tastes that were cued. Specifically, the P1 amplitudes for invalidly cued low-sweet tastes were similar to those observed for the validly cued high-sweet tastes; while amplitudes for the invalidly cued high-sweet tastes were comparable with those observed for the validly cued low-sweet tastes. These findings are critical as they strongly suggest that

expectations are modifying taste perception at early perceptual processing stages as suggested from findings within fMRI research (e.g., Nitschke et al., 2006; Sarinopoulos et al., 2006; Woods et al., 2011). Moreover, similar assimilation effects could be observed within alpha-band oscillations, previously observed for the processing of sweet tastes (Chapters Three and Four). Alpha-ERD for high-sweet tastes was decreased when participants were expecting a low-sweet taste, while alpha-ERD for low-sweet tastes increased when participants were expecting a high-sweet taste.

We also found evidence that taste expectancy modulates the N400 ERP component and reduces activations within the PGC. As discussed in Chapter Five, the N400 ERP is most commonly observed for semantic language anomalies (e.g., Kutas & Federmeier, 2011). However, enhanced N400s have recently been observed for incongruent auditory (e.g., Painter & Koelsch, 2011), visual (e.g., Proverbio & Riva, 2009) and olfactory (Kowalewski & Murphy, 2012) stimulation. For the first time, this ERP component can be extended to primary gustatory processing, further highlighting that this component may function as a general incongruency detector (Kutas & Federmeier, 2011).

In relation to our previous findings, we also observed that high-sweet and low-sweet tastes also differentially affected ERP amplitudes, activations from the PGC and beta-ERD; with high-sweet tastes evoking greater responses from all components. In Chapters Three and Four, we found evidence that early ERP and beta-band oscillations were associated with the processing of taste intensity and the findings from Chapter Five corroborate these interpretations. Furthermore, we confirmed that the early ERP responses to taste intensity were generated by the insula within the PGC, supporting evidence of this regions involvement in taste intensity processing (e.g., Ohla et al., 2010).

Lastly, in Chapter Five, we were able to observe fully articulated ERP peaks. Compared with Chapters Three and Four, we included a reduced taste stimulus set (two tastants), with increased stimuli repetitions (50 repeats) and a decreased testing duration (~ 1 hour). The combination of these factors allowed for a decrease in noise components and the ability achieve a good summation of the evoked potentials over trials.

Limitations

As discussed in section 6.1, the pleasantness ratings obtained in Chapter Five were different to those from our preliminary investigation and were also gradually reduced over the course of the experiment. While not necessarily a limitation for our study, which examined the influence of expectancy on sweetness ratings, this could be a limitation for future investigations; specifically those examining neural responses to taste hedonicity. The quantity of stimulus repeats over a single session allowed for us to obtain articulated ERP components which otherwise may have been obscured by EEG differences that can arise as a result of multiple testing sessions (e.g., Luck, 2005). Therefore, future studies need to weigh the need for good quality EEG data against the need for stabilised taste ratings. It is also possible, however, that the addition of a cognitive component (i.e., expectancy) led to attentional affects that may have contributed to the articulated ERP response. As discussed in Section 5.5; future studies could examine this possibility by determining whether the same ERP quality can be observed when limiting the quantity of trials.

We did not record responses to the anticipatory cues; rather we were interested in the neural responses in relation to the onset of the tastants. Inclusion of this analysis may have informed us on the nature of the processing of anticipation and how this contributed to the effects observed within the EEG data. Thus, such analyses should be included in future studies. Despite this, we were able to gain valuable information pertaining to the effects of cue-elicited expectation on the processing of tastes.

Implications

The implications of these findings are extensive. Most importantly, our results suggest that top-down information influences taste processing at a very early perceptual stage and in primary sensory processing regions, thus implying that the experience of taste is altered by this information. This possibility has been alluded to in fMRI investigations showing altered PGC responses to tastes that have been invalidly cued (Nitschke et al., 2006; Sarinopoulos et al., 2006; Woods et al., 2011). However, processing in primary sensory regions does not necessarily equate to early sensory processing (e.g., Sadacca et al., 2012). For the first time, we demonstrate that expectancies can alter taste processing as early as 100 ms after stimulus onset.

Furthermore, we show that this effect can be bi-directional, in that invalidly cueing both increased and decreased intensity of tastes shifts the behavioural and neural responses towards those that would be engendered were the taste to match the cue.

Such findings have important implications for understanding of the role of extrinsic cues involved in food choice and experience (e.g., Davidenko et al., 2015). For example, why foods labelled as low-fat are often reported as having inferior sensory qualities (e.g., Light et al., 1992), why beverages with a higher-price are rated as more pleasant (Plassmann et al., 2008) and why branded foods and beverages are rated as more palatable than unbranded products (e.g., McClure et al., 2004). Our results suggest that this prior information may be affecting the experience of the tastes consumed. Future research would greatly benefit from the examination of the type of information (i.e., food descriptions, price cues, brand information) that can affect early taste processing mechanisms.

In this study we examined how the expectancy of sweetness alters evaluations and neural responses to sweet tastes. This could be extended to examine changes in hedonic evaluations and neural responses, explored simply by employing cues of hedonicity, similar to those employed for intensity in this thesis, or with more complex cues such as brand information. Similarly, examinations of the expected caloric content of tastes could be conducted to determine if this information alters subjective ratings (e.g., Light et al., 1992) as well as early neural responses to tastants. It might also be useful to explore if people within different BMI categories show differential neural responses to tastants when expectations are manipulated. This may help to determine if there are any underlying neural processing differences in the obese population that may differentially influence their responses to tastes and thus food choices in general. The knowledge gained from studies such as these could help understand how prior beliefs and expectations influence peoples experiences of taste and food and could help determine methods to alter expectations in a way that could encourage healthier food choices (Davidenko et al., 2015). Such methods could include specific cognitive training to promote positive associations (or unlearn negative associations) with healthy foods, a method showing promise within substance use disorders (see Blankers, Salemink & Wiers, 2016 for a review). Moreover, the effectiveness of such methods could be examined longitudinally by monitoring changes in both behavioural and neural responses over time.

It would also be beneficial to examine the extent to which the discrepancy between the expected and actual taste alters perception and processing. In Chapter Five we ensured that while the tastes significantly differed in intensity (as was confirmed by expected and actual ratings), they were not so different in quality or pleasantness as to invoke any substantial contrast effect (Wilson & Klaren, 1992). It would be interesting to determine how much disparity is necessary for contrast effects to occur and what the resulting neural processing changes would be in such cases. This could also help us to understand how information could be used to promote healthier food choices without resulting in contrast effects that could counteract these attempts.

Lastly, future studies should explore the EEG components associated with anticipatory cues, and whether these predict the resulting responses to the tastants. Sarinopoulolos et al. (2006) reported that increased activations in the ACC and OFC during anticipation of a taste following a misleading cue predicted decreased activations in the PGC. EEG investigations could determine whether there are temporal neural predictors of taste expectation effect, thus adding some predictive value to the findings and furthering our understanding of the cortical processes involved in taste expectations.

6.7. Overall Conclusions

This thesis sought to examine central processing of taste and the influence of physiological and psychological factors. To do this we developed a taste stimulus set that could reliably distinguish between taste quality, intensity and hedonicity, as well as a gustometer mechanism that could deliver tastants with temporal precision. We employed various EEG methodologies, including ERP analysis, ERP source localisation and ERD/S analysis and explored the differential coding of taste quality, intensity and hedonicity; how this coding is affected by states of hunger and satiety and the influence of expectancy on taste coding mechanisms. We found that by employing a carefully designed stimulus set with ample repeats, limited testing durations and with taste delivered by a mechanism with temporal precision; good quality gustatory EEG data could be obtained and important observations regarding the coding of taste were determined.

It was found across all studies that ERP analysis was able to determine, to some extent, taste intensity processing within early amplitude increases and hedonicity processing during later ERP processing epochs. In particular, however, it was the ERD/S and source-localisation analysis that was able to better discriminate between tastant characteristics. Beta-band ERD showed consistent increases for more intense tastes, whereas left-lateralised alpha-band ERD was consistently observed for pleasant, sweet tastes. The right insula cortex within the PGC also showed increased activations to stronger taste intensities in one investigation and pleasant tastes resulted in increased activations within the inferior parietal lobule. Therefore, these mechanisms may be critical to taste discrimination processes. Taste quality coding, on the other hand, was indeterminable in all EEG analyses; with responses showing only that flavoured tastes are differentially processed from neutral, water tastes. Taste quality processing appears to be complex and may involve a combination of spatial and temporal patterns that may be better explored using other neuroimaging techniques such as high-density fMRI analysis and intra-cranial recordings (Crouzet et al., 2015).

Hunger and satiety can greatly influence the processing of tastes. In general, hunger enhanced ERP responses and beta-band ERS to all tastants. Sweet tastes evoked differential processing when hungry and sated; eliciting greater ERP amplitudes when sated, but decreased alpha-ERD when hungry. These results suggest that being hungry modifies how the brain attends to and evaluates tastants, particularly in relation to pleasant, nutrient rich tastes. This has important implications for how people make food choices and evaluations under these conditions, and should be evaluated for different BMI groups to determine processing differences that may account for variations in eating behaviours between these groups.

Top-down expectancy information modified the perception and processing of tastants, with responses to unexpected tastes assimilating to the responses that were engendered by tastes that were expected. For the first time, we demonstrated that taste expectancy effects not only occur in primary sensory processing regions (as observed in; Nitschke et al., 2006; Sarinopoulos et al., 2006; Woods et al., 2011) but also during very early perceptual processing stages (~ 100 ms). These results imply that prior information can alter the experience of taste and thus have important

implications for understanding people's experiences of taste and methods to encourage healthier food choices (Davidenko et al., 2015).

Overall, gustation is a complex mechanism that must encode the characteristics of a taste amongst a background of other information sources including current appetite level and prior experience and expectations. This information is instantaneously evaluated by our gustatory system allowing for quick decisions on food selection. The experiments described in this thesis go some way to extend the characterisation of the temporal aspects of this coding and highlight previously unidentified mechanisms that play a role in the sensory perception and evaluation of tastes.

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Appendices

Appendix A: Participant Screening Questionnaire

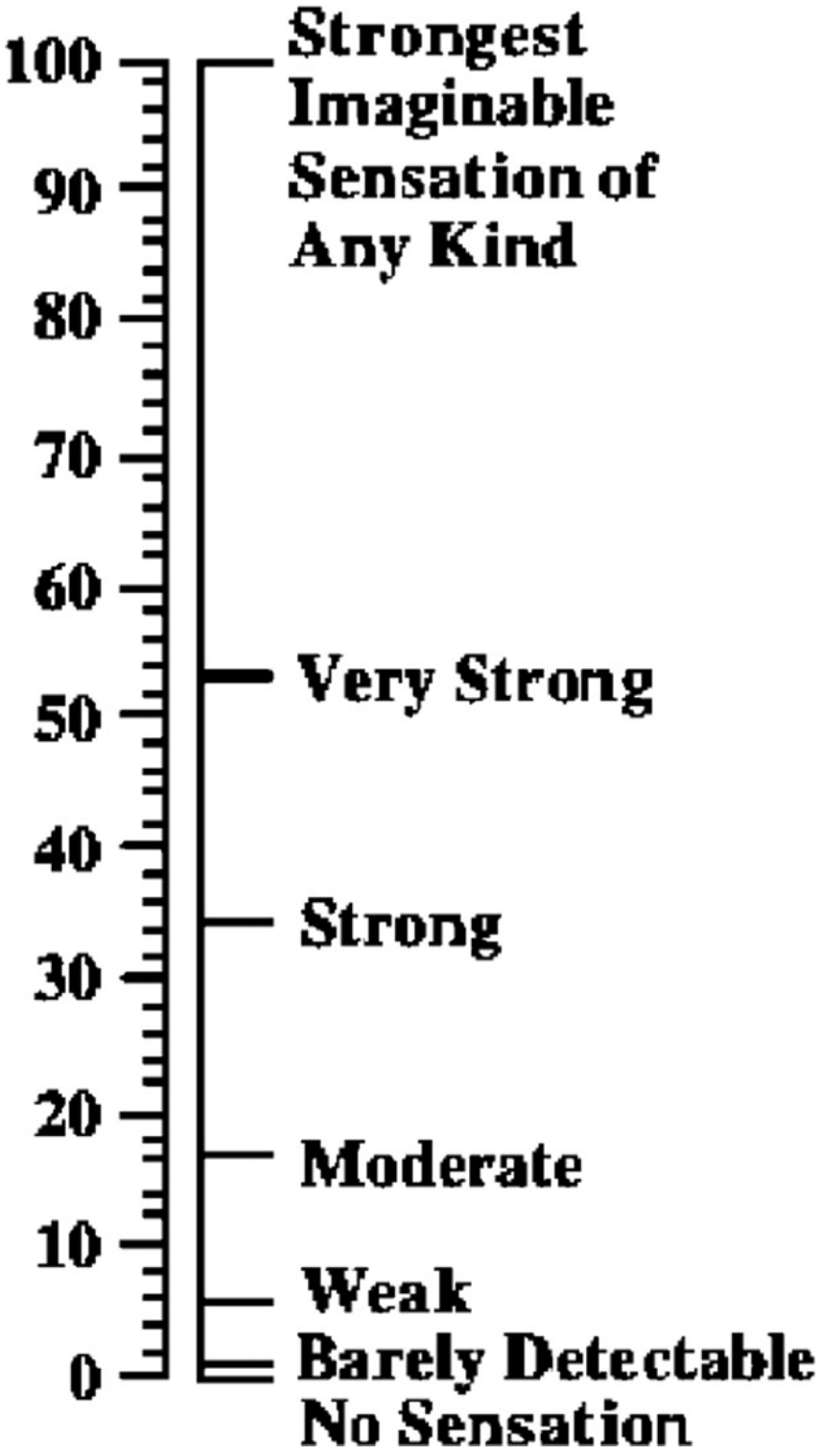
Participant number: ___Age: ___Gender:__Weight:_____Height: _____

Please complete this questionnaire. It collects information that relates to your taste perception and will be used to determine your eligibility for the study. Your results will remain confidential and you have the right to withdraw at anytime and ask for your data to be destroyed

Q1	Do you have any specific allergies or intolerances to the following:	
	Salt (sodium chloride)	○YES ○NO
	Sugar (sucrose)	○YES ○NO
	Quinine (commonly found in tonic water)	○YES ○NO
Q2	Do you have any health condition which may that be affected by ingestion of:	
	Salt	○YES ○NO
	Sugar	○YES ○NO
	Quinine	○YES ○NO
Q3	Are you currently suffering from a condition that may affect your sense of taste or smell? *(Such conditions may include a cold or flu, broken or damaged nose, hay fever or oral injury)* **Please inform us at any time during the study if you are suffering from any such condition as your testing dates will need to be re-arranged*	○YES ○NO
Q4	Are you currently taking any medication that may affect your ability to taste normally? *(Medications*	○YES ○NO

	that can affect your taste ability include; antihistamines, chemotherapy agents, antibiotics, antidepressants and blood pressure medication)	
Q5	Are you a regular smoker?	○YES ○NO
Q8. Please use this space to tell us of anything you think may be important in relation to your sense of taste sense that hasn't been identified above. Any problems you may have with tasting salt, sweet or bitter solutions, or any other allergies which may be relevant to this study		

Appendix B: General Labelled Magnitude Scale (from Bartoshuk et al., 2004)



Appendix C: Labelled Affective Magnitude Scale, adapted from Schutz and Cardello (2004)

Appendix D: Appetite Scale (Flint et al., 2000; Rolls et al., 1999).

Participant number: **Age:** **Height:** **Weight**:

HUNGER QUESTIONNAIRE

INSTRUCTIONS FOR PARTICIPANTS:

Please read each question and then put a mark through the line that best represents how you are feeling in relation to that particular sensation at this moment.

EXAMPLE:
How **TIRED** do you feel **at this moment?**

Not at all tired ——/—— Extremely tired

PLEASE ANSWER THE FOLLOWING QUESTIONS:

How **HUNGRY** do you feel **at this moment**?

Not at all hungry ——————— Extremely hungry

How **FULL** do you feel **at this moment**?

Not at all full ——————— Extremely full

How **SATISFIED** do you feel **at this moment**?

Not at all satisfied _____ Extremely satisfied

How **STRONG** is your desire to **eat at this moment**?

Not at all strong _____ Extremely strong

How **MUCH FOOD** do you feel you could eat **at this moment**?

None at all _____ A large amount

How **THIRSTY** do you feel **at this moment**?

Not at all thirsty _____ Extremely thirsty

How **NAUSEOUS** do you feel **at this moment**?

Not at all nauseous _____ Extremely nauseous

THANK YOU

Appendix E: Gustometer Program

Pump identification

In order to ensure that the system is complete and functional, the program must check for the presence of all pumps at the start of the session. In order to do this, each pump must have an address (1 – 8) installed manually through the program inbuilt into the pump. Each pump address corresponds to a character ['#', ' ', '!', '&','\', '$','%%','*'] when communicating with the computer software. The software sends a serial signal to each pump at the beginning of each testing session to ensure its presence and feedback communication, and to communicate the correct flow rate and duration.

e.g.,

```
symbols = ['#', ' ', '!', '&','\', '$','%%','*'] # checksum symbols for pump communication
strPump = str(pump)
symbol = symbols[int(pump)-1]
 s.write('\2'+'0'+strPump+'KY1'+'\3'+symbol+'\r')
s.readline()
s.write('\2'+'0'+strPump+'KY1'+'\3'+symbol+'\r')
s.readline()
s.write('T'+strPump+'\r')
s.write('\2'+'0'+strPump+'RR10000'+'\3'+strPump+'\r')  # flow rate and  duration indicated here
s.readline()
```

Events & triggers

Trigger events are codes sent to the parallel port of a computer. In EEG studies, triggers need to be precisely simultaneous with the onset of an experimental event I order to accurately determine temporal processing. In this case, the onset of the taste stimuli needed to be coordinated with a parallel port trigger to the EEG data on a

separate computer. To achieve this, the Psychopy program was designed to send a parallel port trigger to the online EEG data, indicating each taste condition (corresponding to a pump address) when it received a returning signal from the selected pump.

e.g.,

```
s.write('\2'+'0'+strPump+'RR10000'+'\3'+strPump+'\r')
 s.readline()
parallel.setData(condition)
```

Randomisation

Trialbooks for each experiment were created within Excel with columns detailing the taste, the condition number and the pump number required to initiate the taste. The Psychopy program was designed to read the Excel file and import these details to the experimental script where appropriate. Randomisation of the trials was achieved by creating a trialbook and either (a) randomising the trial order using the Excel RAND function and creating a sequential trial loop within Psychopy (experiment 1 and 2) or (b) creating a random trial sequence within Psychopy (experiment 3).

e.g.,

```
trials=data.TrialHandler(nReps=1, method='sequential',
trialList=data.importConditions('trialbook1.xlsx'))
trials=data.TrialHandler(nReps=1, method='random',
trialList=data.importConditions('trialbook1.xlsx'))
```

Manual initiation of trials

We needed to be able to control the onset of the stimulus in order that it wasn't concomitant with muscle movements associated with swallowing. To achieve this we added a manual trial initiation feature to the program sequence. This ensured that a trial could only be initiated when the experimenter could be sure that no unwanted movements were being made. This was achieved by the researcher monitoring EMG and video footage of the participant, and pressing 'space' when the taste onset was appropriate.

e.g.,

```
wait.draw()

myWin.flip()

event.waitKeys(keyList=['space', 'escape']) # start pump
stimOut=[pump_number]  # pump started

startPump(stimOut[0],2, condition)
```

Pump failure

Unfortunately, the KNF Stepdos pumps (KNF Verder, Vleuten, The Netherlands) sometimes do not respond to initiation signals, or shut down entirely during the course of the experiment. While this is generally solved by the very technical solution of 'switching off and on again'; it does create problems within the data. The EEG system would still receive a trigger indicating the condition of the trial that was meant to have occurred. In order to avoid including trials in the analysis where the pump failed to pump, we added a trial check procedure into the program. This involved a period several seconds after the taste onset where the experimenter viewed a small dot on the screen which prompted them to enter a response of whether the trial was good (g) or bad (b). When a bad trial was indicated it created a trigger within the EEG data (255) that meant that when cleaning the data offline, the experimenter could discard all trials that preceded a 255 event. The triggers were also included in the Psychopy output file so that behavioral data could be filtered to only include good (g) trials. Moreover, when a bad trial was indicated, the rest of trial sequence was not completed and a new trial was started.

e.g.,

```
checkTrial.draw()

myWin.flip()

trialQuality = event.waitKeys(keyList=['g','b'])

if trialQuality == ['b']:

parallel.setData(255)

trials.addData('aDataQuality',trialQuality)

blankScreen.draw()
```

```
myWin.flip()
core.wait(3.0)
else:
```